EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	2	("6204244").PN.	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	OFF	2006/03/30 13:01
L2	2	("6281244").PN.	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	OFF	2006/03/30 13:01
S1	10	"5656608"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 11:27
S2	9	"5731290"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 11:26
S3	7	"5902829"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 11:27
S4	3	"6013273"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 11:59
S5	2484885	wo "9118610"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 11:59
S6	0	(wo9118610)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 12:00
S7	0	WO9118610	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 12:10

EAST Search History

S8	1256196	green tea extract	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 15:53
S9	8953852	no donor or nitric oxide donor	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 15:54
S11	1824896	nitric oxide donor	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 15:55
S12	65759	S11 same S8	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:00
S13	3808	catechin	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 15:57
S14	299	S12 and S13	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:03
S15	2138702	green tea extract$3 same nitric oxide	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:04
S16	2209760	green tea extract$3 same nitric oxide donor	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:04
S17	2205011	green tea extract$3 near nitric oxide donor	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:17
S18	22934	(green tea extract$3) near9 (nitric oxide donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:09

EAST Search History

S19	4296	catechin or epicatechin or epigallocatechin or gallocatechin	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:17
S20	88	S18 and S19	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:09
S21	4288	catechin or epicatechin or epigallocatechin or gallocatechin and (nitric oxide)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:18
S22	4289	catechin or epicatechin or epigallocatechin or gallocatechin and (nitric oxide donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/29 16:18
S23	0	(green w tea w extract) near9 (nitric w oxide w donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	ADJ	ON	2006/03/29 16:22
S24	0	(green tea extract) same(nitric oxide donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	ADJ	ON	2006/03/29 16:23
S25	1	(green tea extract) same(nitric oxide donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/29 16:23
S26	1	(green tea extract) same(nitric donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/29 16:24
S27	4	(green tea extract) same(nitric donor or NO donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/29 16:25
S28	1194	(green tea extract) and (nitric donor or NO donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/29 16:25

EAST Search History

S29	1194	(green tea extract) and (nitric oxide donor or NO donor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/29 16:25
S30	71266	(green tea or green tea extract$3) and (surgery or surgical)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/30 08:23
S31	8958658	no donor or nitric oxide donor	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/30 08:16
S32	68469	S30 and S31	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 08:16
S33	0	(green tea near9 extract$3) near9 (surgical procedure or surgery)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 08:17
S34	233	(green tea near9 extract$3) and (surgical procedure or surgery)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 08:39
S35	20839	(green tea or green tea extract$3) and (surgery or surgical)and (ntiric oxide)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/30 08:24
S36	1432	(green tea or green tea extract$3) same (surgery or surgical)and (ntiric oxide)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	ON	2006/03/30 08:24
S37	0	(green tea or green tea extract$3) same (surgery or surgical)and (ntiric oxide)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 08:24
S38	1317	(green tea near9 extract$3) and (amino acid or amino acid precursor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 08:40

EAST Search History

S39	252	(green tea near9 extract$3) same (amino acid or amino acid precursor)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 10:28
S40	0	(green tea near9 extract$3) same (ischemia repurfusion)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 10:29
S41	0	(green tea near9 extract$3) and (ischemia repurfusion)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 10:29
S42	2	(green tea) and (ischemia repurfusion)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 10:29
S43	570	(green tea) and (ischemia)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 10:29
S44	77	(green tea near9 extract$3) and (ischemia)	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2006/03/30 12:57

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NEWS 15	FEB 27	New STN AnaVist pricing effective March 1, 2006
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NEWS 17	FEB 28	TOXCENTER reloaded with enhancements
NEWS 18	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 19	MAR 01	INSPEC reloaded and enhanced
NEWS 20	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
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FILE 'HOME' ENTERED AT 13:14:11 ON 30 MAR 2006

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
       AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
       CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
       DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 13:14:32 ON 30 MAR 2006

70 FILES IN THE FILE LIST IN STNINDEX

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=> s (green tea extract###) and ischaemia reperfusion
           2   FILE BIOSIS
           4   FILE CABA
  22 FILES SEARCHED...
           1   FILE EMBASE
           1   FILE ESBIOBASE
           1   FILE MEDLINE
           1   FILE PASCAL
  50 FILES SEARCHED...
           1   FILE SCISEARCH
  69 FILES SEARCHED...

   7 FILES HAVE ONE OR MORE ANSWERS,   70 FILES SEARCHED IN STNINDEX

L1   QUE (GREEN TEA EXTRACT###) AND ISCHAEMIA REPERFUSION

=> s (green tea extract###) and ischemia reperfusion
           1   FILE AGRICOLA
          10   FILE BIOSIS
           4   FILE CABA
          13   FILE CAPLUS
           3   FILE DDFU
           3   FILE DRUGU
          11   FILE EMBASE
           7   FILE ESBIOBASE
  33 FILES SEARCHED...
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           2   FILE LIFESCI
           8   FILE MEDLINE
           6   FILE PASCAL
          10   FILE SCISEARCH
           8   FILE TOXCENTER
          14   FILE USPATFULL
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L2   QUE (GREEN TEA EXTRACT###) AND ISCHEMIA REPERFUSION

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COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
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=> s (green tea extract###) and ischemia reperfusion
        250222 GREEN
          2406 GREENS
        251551 GREEN
                 (GREEN OR GREENS)
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         33862 TEA
                 (TEA OR TEAS)
        250253 EXTRACT###
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        223333 EXTS
        470842 EXT
                 (EXT OR EXTS)
        352214 EXTD
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        352216 EXTD
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             1 EXTGS
         54841 EXTG
                 (EXTG OR EXTGS)
        391734 EXTN
         14061 EXTNS
        397260 EXTN
                 (EXTN OR EXTNS)
       1065363 EXTRACT###
                 (EXTRACT### OR EXT OR EXTD OR EXTG OR EXTN)
          1037 GREEN TEA EXTRACT###
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         66759 ISCHEMIA
            71 ISCHEMIAS
         66774 ISCHEMIA
                 (ISCHEMIA OR ISCHEMIAS)
         28274 REPERFUSION
            52 REPERFUSIONS
         28283 REPERFUSION
                 (REPERFUSION OR REPERFUSIONS)
         14410 ISCHEMIA REPERFUSION
                 (ISCHEMIA(W)REPERFUSION)
L3          13 (GREEN TEA EXTRACT###) AND ISCHEMIA REPERFUSION
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=> d total ibib abs

L3 ANSWER 1 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1101390 CAPLUS
DOCUMENT NUMBER: 144:535
TITLE: Inhibitory effect of **green tea extract** on β-amyloid-induced PC12 cell death by inhibition of the activation of NF-κB and ERK/p38 MAP kinase pathway through antioxidant mechanisms
AUTHOR(S): Lee, Sun Young; Lee, Jae Woong; Lee, Heesoon; Yoo, Han Soo; Yun, Yeo Pyo; Oh, Ki Wan; Ha, Tae Youl; Hong, Jin Tae
CORPORATE SOURCE: College of Pharmacy, Chungbuk National University, Chungbuk, Cheongju, Heungduk-gu, 361-763, S. Korea
SOURCE: Molecular Brain Research (2005), 140(1-2), 45-54
CODEN: MBREE4; ISSN: 0169-328X
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Beta-amyloid peptide (Aβ) is considered responsible for the pathogenesis of Alzheimer's disease (AD). Several lines of evidence support that Aβ-induced cytotoxicity is mediated through the generation of reactive oxygen species (ROS). Thus, agents that scavenge ROS level may usefully impede the development or progress of AD. **Green tea extract** has been known to have such antioxidant properties. Our previous studies demonstrate that **green tea extract** protected **ischemia/reperfusion**-induced brain cell death by scavenging oxidative damages of macromols. In this study, we investigated the effects of **green tea extract** on Aβ-induced oxidative cell death in cultured rat pheochromocytoma (PC12) cells. PC12 cells treated with Aβ25-35 (10-50 μM) showed intracellular ROS elevation, the formation of 8-oxodG (an oxidized form of DNA), and underwent apoptotic cell death in a dose-dependent manner. Aβ25-35 treatment upregulated pro-apoptotic p53 at the gene level, and Bax and caspase-3 at the protein level, but downregulated anti-apoptotic Bcl-2 protein. Interestingly, co-treated **green tea extract** (10-50 μg/mL) dose-dependently attenuated Aβ25-35 (50 μM)-induced cell death, intracellular ROS levels, and 8-oxodG formation, in addition to p53, Bax, and caspase-3 expression, but upregulated Bcl-2. Furthermore, **green tea extract** prevented the Aβ25-35-induced activations of the NF-κB and ERK and p38 MAP kinase pathways. Our study suggests that **green tea extract** may usefully prevent or retard the development and progression of AD.
REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:691126 CAPLUS
DOCUMENT NUMBER: 143:318846
TITLE: Green tea polyphenol extract attenuates **ischemia/reperfusion** injury of the gut
AUTHOR(S): Muia, Carmelo; Mazzon, Emanuela; Paola, Rosanna; Genovese, Tiziana; Menegazzi, Marta; Caputi, Achille P.; Suzuki, Hisanori; Cuzzocrea, Salvatore
CORPORATE SOURCE: Department of Clinical and Experimental Medicine and Pharmacology, Torre Biologica, Policlinico Universitario, Messina, 98123, Italy
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology (2005), 371(5), 364-374
CODEN: NSAPCC; ISSN: 0028-1298

PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Various studies have clearly demonstrated that green tea catechins possess potent antioxidative properties, and the preventive effects against various oxidative diseases have been reported. The aim of this study was to investigate the effect of **green tea extract** on the tissue injury caused by **ischemia/reperfusion** (I/R) of the gut. I/R injury of the intestine was caused by clamping both the superior mesenteric artery and the celiac trunk for 45 min followed by release of the clamp allowing reperfusion for 1 h or 4 h. This procedure results in splanchnic artery occlusion (SAO) shock. Rats subjected to SAO developed a significant fall in mean arterial blood pressure, and only 10% of the animals survived for the entire 4-h reperfusion period. Surviving animals were sacrificed for histol. examination and biochem. studies. Rats subjected to SAO displayed a significant increase in tissue myeloperoxidase (MPO) activity and malondialdehyde (MDA) levels, significant increases in plasma tumor necrosis factor (TNF)-α levels and marked injury to the distal ileum. Increased immunoreactivity to nitrotyrosine was observed in the ileum of rats subjected to SAO. Staining of sections of the ileum obtained from SAO rats with anti-intercellular adhesion mol. (ICAM-1) antibody and with anti-P-selectin antibody resulted in diffuse staining. Administration of **green tea extract** (20 and 10 mg kg-1 i.v.) 15 min prior to the onset of gut reperfusion significantly reduced in a dose-dependent manner the fall in mean arterial blood pressure, the mortality rate, infiltration of the reperfused intestine with polymorphonuclear neutrophils (MPO activity), lipid peroxidn. (MDA levels), production of TNF-α, and histol. evidence of gut injury. Administration of **green tea extract** also markedly reduced nitrotyrosine formation and the up-regulation of ICAM-1 and P-selectin during reperfusion. To clarify that **green tea extract** might be useful in the therapy of I/R injury, we also investigated the effect of **green tea extract** (20 mg kg-1 i.v.) when administered 5 min after the onset of gut reperfusion. Similar to the pretreatment approach, the post-treatment also significantly reduced the gut injury induced by I/R. These results demonstrate that **green tea ext** . significantly reduces I/R injury of the intestine.
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:634307 CAPLUS
DOCUMENT NUMBER: 143:259192
TITLE: STAT1 as a new molecular target of anti-inflammatory treatment
AUTHOR(S): Carcereri de Prati, Alessandra; Ciampa, Anna Rosa; Cavalieri, Elisabetta; Zaffini, Raffaela; Darra, Elena; Menegazzi, Marta; Suzuki, Hisanori; Mariotto, Sofia
CORPORATE SOURCE: Section of Biochemistry, Department of Neuroscience and Vision, University of Verona, Verona, 37134, Italy
SOURCE: Current Medicinal Chemistry (2005), 12(16), 1819-1828
CODEN: CMCHE7; ISSN: 0929-8673
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Cyclooxygenase (COX) is widely considered as the mol. target of non-steroid anti-inflammatory drugs (NSAIDs). However, due to the harmful side effect frequently observed following chronic use, the development of new anti-inflammatory agents is the matter of many studies. Signal transducers and activators of transcription (STAT) are a family of nuclear proteins mediating the action of a number of cytokines. Among them, STAT1 plays a critical role in the signal transduction pathway of interferon-gamma (IFN-gamma) and growth hormone. STAT1 cascade is one major signaling

pathway converting the IFN-gamma signal into gene expression, such as inducible nitric oxide synthase (iNOS), COX, vascular cell adhesion mols. (VCAM) and intercellular cell adhesion mols. (ICAM), critically involved in different pathologies correlated to the inflammatory process. This review focuses the attention on an alternative approach to the development of novel drugs based on inhibition of STAT1 pathway. In this context, a growing interest has been focused on natural compds. We have recently reported a several data indicating that **green tea extract** (GTE), St. John's Wort extract and epigallocatechin-3-gallate (EGCG) exhibit a specific and strong anti-STAT1 activity which is independent of their acclaimed strong anti-oxidant activity. More recently, GTE has been shown to protect heart damage from **ischemia/reperfusion** in rats, suggesting that the protective effect of green tea might be correlated to its anti-STAT1 activity. The present review is aimed at providing data that STAT1 may potentially be claimed as a new mol. target of an anti-inflammatory treatment and that among natural compds. there are a number of anti-STAT1 substances.

REFERENCE COUNT: 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:815203 CAPLUS

DOCUMENT NUMBER: 141:288837

TITLE: Epigallocatechin-3-gallate inhibits STAT1 activation and protects cardiac myocytes from **ischemia/reperfusion**-induced apoptosis

AUTHOR(S): Townsend, Paul A.; Scarabelli, Tiziano M.; Pasini, Evasio; Gitti, Gianluca; Menegazzi, Marta; Suzuki, Hisanori; Knight, Richard A.; Latchman, David S.; Stephanou, Anastasis

CORPORATE SOURCE: Medical Molecular Biology Unit, Institute of Child Health, University College London, London, WC1N 1EH, UK

SOURCE: FASEB Journal (2004), 18(13), 1621-1623, 10.1096/fj.04-1716fje
CODEN: FAJOEC; ISSN: 0892-6638

PUBLISHER: Federation of American Societies for Experimental Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors have previously demonstrated that STAT-1 plays a critical role in promoting apoptotic cell death in cardiac myocytes following **ischemia/reperfusion** (I/R) injury. Epigallocatechin-3-gallate (EGCG), the major constituent of green tea, has recently been reported to inhibit STAT-1 activity in noncardiac cells. In the present study, the authors have assessed the protective effects of EGCG and **green tea extract** (GTE) infusion on both cultures of cardiac myocytes and the isolated rat heart. EGCG reduced STAT-1 phosphorylation and protected cardiac myocytes against I/R-induced apoptotic cell death. Moreover, EGCG reduced the expression of a known STAT-1 pro-apoptotic target gene, Fas receptor. More interestingly, oral administration of GTE as well as EGCG infusion limited the extent of infarct size and attenuated the magnitude of myocyte apoptosis in the isolated rat heart exposed to I/R injury. This reduction cell death was associated with improved hemodynamic recovery and ventricular function in the ischemic/reperfused rat heart. This is the first report to show that consumption of green tea is able to mediate cardioprotection and enhance cardiac function during I/R injury. Because GTE-mediated cardioprotection is achieved, at least in part, through inhibition of STAT-1 activity, the authors may postulate that a similar action can be implemented in the clin. setting to minimize STAT-1 activation levels in patients with acute coronary artery disease (CAD).

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:203590 CAPLUS
DOCUMENT NUMBER: 140:210831
TITLE: Composition for protecting organ, tissue or cell and utilization thereof
INVENTOR(S): Komeda, Masashi; Hyon, Suong-Hyu; Miwa, Senri
PATENT ASSIGNEE(S): MG Pharmacy Inc., Japan
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004019680	A1	20040311	WO 2003-JP11127	20030829
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003261860	A1	20040319	AU 2003-261860	20030829
EP 1535514	A1	20050601	EP 2003-791435	20030829
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.: JP 2002-255979 A 20020830
WO 2003-JP11127 W 20030829

AB It is intended to provide a composition whereby an organ, a tissue or cells can be efficiently protected and preserved, in particular, a protective composition which can be used even during an operation. More specifically, a composition containing polyphenols for protecting and preserving an organ, a tissue or cells is provided. It is also intended to provide a method of protecting an organ, a tissue or cells in a sample which involves the step of exposing the organ, tissue or cells to polyphenol. This composition and polyphenol are efficacious in protecting the functions of an organ (in particular, heart, brain, nerve, spinal cord, etc.). **Green tea exts.** containing epigallocatechin gallate were orally administered to rats and tested for heart-protecting effects during **ischemia/reperfusion.**

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:684340 CAPLUS
DOCUMENT NUMBER: 140:246727
TITLE: Protective effects of the green tea polyphenol, (-)-epigallocatechin gallate against **ischemia reperfusion** injury induced by middle cerebral artery occlusion in rats
AUTHOR(S): Choi, Young-Bin; Park, Jeong-Wook; Han, Si-Ryung; Lee, Kwang-Soo; Kim, Beum-Saeng
CORPORATE SOURCE: Department of Neurology, College of Medicine, The Catholic University of Korea, S. Korea
SOURCE: Taehan Sin'gyong Kwahak Hoechi (2003), 21(4), 387-391
CODEN: TSKHC2; ISSN: 1225-7044
PUBLISHER: Korean Neurological Association
DOCUMENT TYPE: Journal
LANGUAGE: Korean
AB EGCG (epigallocatechin gallate), a major **green tea**

extract, is a potent free radical scavenger which has been shown to reduce free radical-induced lipid peroxidn. The purpose of this study was to examine whether EGCG reduces focal **ischemia/reperfusion**-induced brain injury in rats. Male Wistar rats were anesthetized with ketamine and xylazine and subjected to 120 min of temporary middle cerebral artery occlusion by an intraluminal nylon suture coated with poly-L-lysine. The drug (EGCG, n=8) or vehicle (normal saline, n=8) was administered i.v.(as a 50 mg/kg bolus) immediately after the onset of middle cerebral artery occlusion. Neurol. status was evaluated 2 h after occlusion and 24 h after. Twenty-four hours after ischemia, the brain was perfusion-fixated and the infarct volume was determined EGCG significantly improved the neurol. status at 24 h after middle cerebral artery occlusion.($p<0.05$), and reduced total infarct vols. ($p<0.01$). These results demonstrate the neuroprotective effect of EGCG in a rat model of transient focal cerebral ischemia.

L3 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:484552 CAPLUS
DOCUMENT NUMBER: 139:245148
TITLE: Antioxidant nutrients and hypoxia/ischemia brain injury in rodents
AUTHOR(S): Ikeda, Katsumi; Negishi, Hiroko; Yamori, Yukio
CORPORATE SOURCE: School of Human Environmental Sciences, Mukogawa Women's University, Ikebiraki-cho, Nishinomiya, Japan
SOURCE: Toxicology (2003), 189(1-2), 55-61
CODEN: TXCYAC; ISSN: 0300-483X
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Cerebral ischemia and recirculation cause delayed neuronal death in rodents, such as Mongolian gerbils and stroke-prone spontaneously hypertensive rats (SHRSP), used as exptl. stroke models. Enhanced nitric oxide production, occurrence of apoptosis, and attenuated redox regulatory system contribute to the development of delayed neuronal death. Many studies have suggested beneficial effects of antioxidant nutrients, such as vitamin E, **green tea extract**, ginkgo biloba extract, resveratrol and niacin, in cerebral ischemia and recirculation brain injury. These results are important for attenuation of deleterious consequences of oxidative stress in ischemia and recirculation injury.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:126725 CAPLUS
DOCUMENT NUMBER: 139:255093
TITLE: Effect of **green tea extracted** polyphenol on **ischemia/reperfusion** injury after cold preservation of rat lung
AUTHOR(S): Omasa, M.; Fukuse, T.; Matsuoka, K.; Inui, K.; Hyon, S. H.; Wada, H.
CORPORATE SOURCE: Department of Thoracic Surgery, Institute for Frontier Medical Sciences, Kyoto, Japan
SOURCE: Transplantation Proceedings (2003), 35(1), 138-139
CODEN: TRPPA8; ISSN: 0041-1345
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Ischemia/reperfusion** injury (I/R) is the main cause of early graft failure in lung transplantation. Reactive oxygen species (ROS) play critical roles in I/R injury. **Green tea extracted** polyphenol (GTP) is known to have anticancer, antiinflammatory and antioxidant effects. We investigated the influence of GTP on I/R injury after cold preservation of the rat lung using an ex vivo rat lung perfusion circuit. In this experiment, GTP (0.04 to 1.0 mg/mL)

did not ameliorate the early I/R injury after cold preservation of the rat lung. Furthermore, it did not show a dose-escalation effect. However, further testing of GTP should be conducted to investigate the late I/R injury because it may decrease inflammatory cytokine production

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:862167 CAPLUS
DOCUMENT NUMBER: 138:368066
TITLE: Protection of hypoxia/reoxygenation injury by green tea supplementation in cultured cardiac cells
AUTHOR(S): Bordoni, Alessandra; Hrelia, Silvana; Angeloni, Cristina; Leoncini, Emanuela; Giordano, Emanuele; Guarnieri, Carlo; Caldarera, Claudio M.; Biagi, Pier L.
CORPORATE SOURCE: Nutrition Research Center (Department of Biochemistry), Alma Mater Studiorum University of Bologna, Bologna, 40126, Italy
SOURCE: Free Radical Research (2002), 36(Suppl. 1), 75-76
CODEN: FRARER; ISSN: 1071-5762
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The supplementation of green tea in cultured cardiac cells for the protection of hypoxia/reoxygenation injury was studied. Green tea is an excellent source of polyphenol antioxidants known as catechins. Results obtained with **green tea extract** were compared with those obtained with α-tocopherol, since pretreatment with vitamin E has been demonstrated to attenuate **ischemia-reperfusion** injury. Antioxidants demonstrated a striking protective effect, decreasing both LDH release and conjugated diene production; **green tea extract** showed a dose related effect, with a maximum at 50 μg/mL concentration Any intervention that attenuates the severity of the hypoxic injury will also attenuate the severity of the subsequent reoxygenation injury. The administration of antioxidants prior to the onset of ischemia may reduce tissue damage.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:809927 CAPLUS
DOCUMENT NUMBER: 138:348524
TITLE: Prevention of hepatic **ischemia-reperfusion** injury by **green tea extract**
AUTHOR(S): Zhong, Zhi; Froh, Matthias; Connor, Henry D.; Li, Xiangli; Conzelmann, Lars O.; Mason, Ronald P.; Lemasters, John J.; Thurman, Ronald G.
CORPORATE SOURCE: Departments of Cell and Developmental Biology and Pharmacology, University of North Carolina at Chapel Hill, Chapel Hill, NC, 27599, USA
SOURCE: American Journal of Physiology (2002), 283(4, Pt. 1), G957-G964
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB These expts. were designed to determine whether **green tea extract** (GTE), which contains polyphenolic free radical scavengers, prevents **ischemia-reperfusion** injury to the liver. Rats were fed a powdered diet containing 0-0.3% GTE starting 5 days before hepatic warm ischemia and reperfusion. Free radicals in bile were trapped with the spin-trapping reagent α-(4-pyridyl-1-oxide)-N-tert-butylnitrone (4-POBN) and measured using ESR spectroscopy. Hepatic

ischemia-reperfusion increased transaminase release and caused pathol. changes including focal necrosis and hepatic leukocyte infiltration in the liver. Transaminase release was diminished by over 85% and pathol. changes were almost totally blocked by 0.1% dietary GTE. **Ischemia-reperfusion** increased 4-POBN/radical adducts in bile nearly twofold, an effect largely blocked by GTE. Epicatechin, one of the major green tea polyphenols, gave similar protection as GTE. In addition, hepatic **ischemia-reperfusion** activated NF-κB and increased TNF-α mRNA and protein expression. These effects were all blocked by GTE. Taken together, these results demonstrate that GTE scavenges free radicals in the liver after ischemia-reoxygenation, thus preventing formation of toxic cytokines. Therefore, GTE could prove to be effective in decreasing hepatic injury in disease states where **ischemia-reperfusion** occurs.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:113317 CAPLUS

DOCUMENT NUMBER: 135:142037

TITLE: Neuroprotective effect of **green tea extract** in experimental **ischemia-reperfusion** brain injury

AUTHOR(S): Hong, J. T.; Ryu, S. R.; Kim, H. J.; Lee, J. K.; Lee, S. H.; Kim, D. B.; Yun, Y. P.; Ryu, J. H.; Lee, B. M.; Kim, P. Y.

CORPORATE SOURCE: National Institute of Toxicological Research, Korea Food and Drug Administration, Seoul, S. Korea

SOURCE: Brain Research Bulletin (2001), Volume Date 2000, 53(6), 743-749
CODEN: BRBUDU; ISSN: 0361-9230

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Eicosanoids accumulation and formation of O free radicals were implicated in the pathogenesis of **ischemia/reperfusion** brain injury. In the present study, the authors examined whether **green tea extract** protects against **ischemia/reperfusion**-induced brain injury by minimizing eicosanoid accumulation and O radical-induced oxidative damage in the brain. **Green tea extract** (0.5%) was orally administered to Wistar rats for 3 wk before induction of ischemia. Ischemia was induced by the occlusion of middle cerebral arteries for 60 min and reperfusion was achieved for 24 h. Infarction volume in the ipsilateral hemisphere of **ischemia/reperfusion** animals was 114 ± 16 mm3 in the 0.5% green tea pretreated animals compared to 180 ± 54 mm3 in left hemisphere of nontreated animals. **Green tea extract** (0.5%) also reduced **ischemia/reperfusion**-induced eicosanoid concentration: leukotriene C4 (from 245 ± 51 to 186 ± 22), prostaglandin E2 (from 306 ± 71 to 212 ± 43) and thromboxane A2 (327 ± 69 to 251 ± 87 ng/mg protein). **Ischemia/reperfusion**-induced increases of hydrogen peroxide level (from 688 ± 76 to 501 ± 99 nmole/mg protein), lipid peroxidn. products (from 1010 ± 110 to 820 ± 70 nmole/mg protein) and 8-oxodG formation (from 1.3 ± 0.3 to 0.8 ± 0.2 ng/μg DNA, +10-2) were also reduced. Moreover, 0.5% **green tea extract** also reduced the apoptotic cell number (from 44 ± 11 to 29 ± 1 in the striatum, and from 72 ± 11 to 42 ± 5 apoptotic cells/high power field in the cortex region). **Green tea extract** pretreatment also promoted recovery from the **ischemia/reperfusion**-induced inhibition of active avoidance. The present study shows that the minimizing effect of **green tea extract** on the eicosanoid accumulation and oxidative damage in addition to the reduction of neuronal cell death could eventually result in protective effect on the **ischemia/**

reperfusion-induced brain injury and behavior deficit.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:7986 CAPLUS
DOCUMENT NUMBER: 134:236821
TITLE: Protective effect of **green tea** **extract** on **ischemia/** **reperfusion**-induced brain injury in Mongolian gerbils
AUTHOR(S): Hong, J. T.; Ryu, S. R.; Kim, H. J.; Lee, J. K.; Lee, S. H.; Yun, Y. P.; Lee, B. M.; Kim, P. Y.
CORPORATE SOURCE: National Institute of Toxicological Research, Korea Food and Drug Administration, Seoul, Eunpyung-gu, Nokbun-dong, 122-704, S. Korea
SOURCE: Brain Research (2001), 888(1), 11-18
CODEN: BRREAP; ISSN: 0006-8993
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Free radical-induced oxidative damages of macromols. and cell death are important factors in the pathogenesis of **ischemia/** **reperfusion** brain injury. In the present study, an investigation as to whether **green tea extract** reduces **ischemia/reperfusion**-induced brain injury in Mongolian gerbils was conducted. The effect of green tea on the **ischemia/** **reperfusion**-induced production of hydrogen peroxide, lipid peroxidn. and oxidative DNA damage (formation of 8-hydroxydeoxyguanosine), and cell death in addition to locomotor activity was studied. Two doses (0.5 or 2%) of **green tea extract** were added into the drinking water and to be accessed by animals ad libitum for 3 wk prior to the induction of ischemia. A global ischemia was induced by the bilateral occlusion of the common carotid arteries for 5 min. Reperfusion was achieved by releasing the occlusion and restoring blood circulation for 48 h. The infarction vols. were 112±31 mm3 and 76±11 mm3 in the 0.5 and 2% green tea pretreated animals compared to 189±12 mm3 in the **ischemia/reperfusion** animals. **Green** **tea extract** also reduced the levels of **ischemia/** **reperfusion**-induced hydrogen peroxide (from 1470±170 to 1034±46 and 555±30 nmole/mg protein), lipid peroxidn. products (from 1410±210 to 930±40 and 330±20 nmole/mg protein) and 8-oxodG (from 3.9±0.1 to 2.8±0.3 and 1.9±0.3 ng/μg DNA, +10-2) by pretreatment of 0.5 or 2% green tea for 3 wk, resp. Moreover, green tea also reduced the number of **ischemia/reperfusion**-induced apoptotic cells (from 59±12 to 37±8, 15±11 apoptotic cells/high power field in the striatum region) and locomotor activity (from 15140±2940 to 3900±600 and 4100±1200). This study therefore suggests that green tea may be a useful agent for the prevention of cerebral ischemia damage.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:406065 CAPLUS
DOCUMENT NUMBER: 133:163655
TITLE: Protective effect of **green tea** **extract** against reperfusion injury in rats: antioxidant and anti-inflammatory properties
AUTHOR(S): Yagi, Nobuaki; Yoshikawa, Toshikazu; Naito, Yuji; Matsuyama, Kiichi; Tanaka, Yukiko; Ochiai, Jun; Yoshida, Norimasa; Kondo, Motoharu
CORPORATE SOURCE: First Department of Medicine, Kyoto Prefectural University of Medicine, Kyoto, 602-8566, Japan
SOURCE: Journal of Clinical Biochemistry and Nutrition (1999),

27(2), 89-101
CODEN: JCBNER; ISSN: 0912-0009
PUBLISHER: Institute of Applied Biochemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of **green tea extract** on acute gastric mucosal damage induced by **ischemia-reperfusion** injury was investigated in rats. **Ischemia-reperfusion** injury was produced by applying a small vascular clamp to the celiac artery for 30 min followed by removal of the clamp with reperfusion for 60 min. An anti-ulcer effect of the **green tea extract** was demonstrated in this model. The increase seen in the lipid peroxide level in the gastric mucosa after **ischemia-reperfusion** was significantly inhibited by the extract Tissue-associated myeloperoxidase activity, an index of neutrophil accumulation, was increased significantly in the gastric mucosa after reperfusion; this increase of activity was significantly inhibited by the **green tea extract** and paralleled the increase in the total area of gastric erosions. An ESR spin trapping study showed that the extract scavenged superoxide radicals generated by the hypoxanthine-xanthine oxidase system and that diphenyl-p-picryl-hydrazyl radicals were also eliminated in a concentration-dependent manner. In an in vitro study, the **green tea extract** significantly inhibited the increase in lipid peroxide in brain homogenates. Incubation of whole blood cells with interleukin-8 increased the expression of CD11b/CD18 by neutrophils, whereas co-incubation with the extract did not cause this upregulation. Human umbilical vein endothelial cells stimulated with interleukin-1β showed increased expression of E-selectin and intercellular adhesion mol.-1, but co-incubation with the extract significantly inhibited this upregulation. These results suggest that the protective effect of **green tea extract** against **ischemia/reperfusion** -induced gastric mucosal injury may be related to its antioxidant activity and inhibition of neutrophil accumulation.
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

```
=> s arginine and ischemia reperfusion
        109204 ARGININE
          1068 ARGININES
        109561 ARGININE
                 (ARGININE OR ARGININES)
         66759 ISCHEMIA
            71 ISCHEMIAS
         66774 ISCHEMIA
                 (ISCHEMIA OR ISCHEMIAS)
         28274 REPERFUSION
            52 REPERFUSIONS
         28283 REPERFUSION
                 (REPERFUSION OR REPERFUSIONS)
         14410 ISCHEMIA REPERFUSION
                 (ISCHEMIA(W)REPERFUSION)
L4         557 ARGININE AND ISCHEMIA REPERFUSION

=> dup rem
ENTER L# LIST OR (END):l4
PROCESSING COMPLETED FOR L4
L5          557 DUP REM L4 (0 DUPLICATES REMOVED)

=> d scan

L5    557 ANSWERS   CAPLUS  COPYRIGHT 2006 ACS on STN
CC   1-8 (Pharmacology)
TI   Oral Administration of Geranylgeranylacetone Blunts the Endothelial
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Dysfunction Induced by Ischemia and Reperfusion in the Rat Heart
ST geranylgeranylacetone heart ischemia cardioprotective mechanism
IT Cytoprotective agents
(cardioprotective; oral administration of geranylgeranylacetone blunts the endothelial dysfunction induced by ischemia and reperfusion in the rat heart)
IT Blood vessel, disease
(endothelium; oral administration of geranylgeranylacetone blunts the endothelial dysfunction induced by ischemia and reperfusion in the rat heart)
IT Heart, disease
(**ischemia-reperfusion** injury; oral administration of geranylgeranylacetone blunts the endothelial dysfunction induced by ischemia and reperfusion in the rat heart)
IT Endothelium
(vascular, disease; oral administration of geranylgeranylacetone blunts the endothelial dysfunction induced by ischemia and reperfusion in the rat heart)
IT 115926-52-8, PI3 kinase 182372-13-0, Rho kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(oral administration of geranylgeranylacetone blunts the endothelial dysfunction induced by ischemia and reperfusion in the rat heart)
IT 6809-52-5, Geranylgeranylacetone
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oral administration of geranylgeranylacetone blunts the endothelial dysfunction induced by ischemia and reperfusion in the rat heart)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> s (arginine and glycine) and ischemia reperfusion
109204 ARGININE
1068 ARGININES
109561 ARGININE
(ARGININE OR ARGININES)
146966 GLYCINE
2035 GLYCINES
147845 GLYCINE
(GLYCINE OR GLYCINES)
66759 ISCHEMIA
71 ISCHEMIAS
66774 ISCHEMIA
(ISCHEMIA OR ISCHEMIAS)
28274 REPERFUSION
52 REPERFUSIONS
28283 REPERFUSION
(REPERFUSION OR REPERFUSIONS)
14410 ISCHEMIA REPERFUSION
(ISCHEMIA(W)REPERFUSION)
L6 11 (ARGININE AND GLYCINE) AND ISCHEMIA REPERFUSION

=> d scan

L6 11 ANSWERS CAPLUS COPYRIGHT 2006 ACS on STN
CC 11-1 (Plant Biochemistry)
Section cross-reference(s): 1, 30
TI Triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root)
ST terpene deriative myrrh olibanum saussurea nitric oxide inhibitor
IT Heat-shock proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HSP 72; triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT Perfumes
(myrrh; triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT Resins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(olibanum; triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT Saussurea
(root; triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT Boswellia carterii
Macrophage
New natural products
Saussurea lappa
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT Progestogens
Sesquiterpenes
Terpenes, biological studies
Triterpenes
RL: PAC (Pharmacological activity); PUR (Purification or recovery); BIOL (Biological study); PREP (Preparation)
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT Natural products, pharmaceutical
RL: PAC (Pharmacological activity); PUR (Purification or recovery); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT 10102-43-9, Nitric oxide, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT 57-83-0P, Progesterone, biological studies 471-16-9P, (+)-Sabinol 545-47-1P, Lupeol 1617-70-5P, Lupenone 2118-90-3P, Neoilexonol 2348-66-5P, Neoilexonol acetate 4031-52-1P, p-Menth-5-ene-1,2-diol 4439-99-0P, Epilupeol 5389-76-4P, Urs-12-ene-3β,11α-diol 6610-54-4P, 3-O-Acetylepilupeol 6610-56-6P, Glochidiol 14351-29-2P, Dammarenediol-II 22558-20-9P, 3-O-Acetyldammarenediol-II 25269-17-4P, Isocembrol 39025-23-5P, 4,17(20)-cis-Pregnadiene-3,6-dione 39025-24-6P, 4,17(20)-(trans)-Pregnadiene-3,16-dione 39025-25-7P, Guggulsterol I 41753-44-0P, Ursa-9(11),12-dien-3β-ol 41943-03-7P, Mukulol 42348-26-5P, 20S-Acetyloxy-4-pregnene-3,16-dione 53342-72-6P, 3-O-Acetylisofouquierol 53822-99-4P, Isofouquierol 61448-03-1P, Lup-20(29)-ene-2α,3β-diol 71697-84-2P, (-)-trans-Sobrerol 80126-41-6P, 4-Epi-Isocembrol 85769-68-2P, Pregn-4-ene-3,16-dione 94415-61-9P, 20R,22R-Dihydroxy-cholest-4-en-3-one 102848-62-4P, Mansumbinol 109795-18-8P, Olibanumol H 126313-88-0P, Urs-12-ene-3α,11α-diol 142790-85-0P, 3β-Hydroxymansumbin-13(17)-ene-16-one 161906-32-7P 227004-15-1P 350809-42-6P, Myrrhanol A 350809-44-8P, Myrrhanone A 359875-83-5P, Olibanumol C 446030-41-7P, Myrrhanol B 446030-42-8P 446030-43-9P, Myrrhanone B 676267-96-2P 676267-97-3P, 3α-Acetoxylup-20(29)-ene-11α-ol 676267-98-4P 676267-99-5P, 11-Methoxy-epi-α-amyrin 676268-00-1P, 3-O-Acetyl-11-methoxy-epi-α-amyrin 676268-01-2P 676327-82-5P, Olibanumol A 676327-83-6P, Olibanumol B 676327-84-7P, Olibanumol D 676327-85-8P, Olibanumol E 676327-86-9P, Olibanumol F

676327-87-0P, Olibanumol G 676327-88-1P, Olibanumol I 676327-89-2P, Olibanumol J 676327-90-5P, Olibanumol K 676327-91-6P, Olibanumol L 676327-92-7P, Olibanumol M 676327-93-8P, 3-O-Acetyl-3β,20S,24S-trihydroxydammar-25-ene 676327-94-9P, 3-O-Acetyl-3β,20S,24R-trihydroxydammar-25-ene
RL: PAC (Pharmacological activity); PUR (Purification or recovery); BIOL (Biological study); PREP (Preparation)
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT 477-43-0P, Dehydrocostus lactone 553-21-9P, Costunolide
RL: PAC (Pharmacological activity); PUR (Purification or recovery); RCT (Reactant); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent)
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT 126209-82-3P, Saussureamine B 148225-51-8P, Saussureamine D 148225-52-9P, Saussureamine E 148245-82-3P, Saussureamine A 148245-83-4P, Saussureamine C 301301-14-4P 308789-77-7P 308789-79-9P 308789-80-2P 308789-81-3P 308789-82-4P 308789-83-5P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

IT 52-90-4, L-Cysteine, reactions 56-40-6, **Glycine**, reactions 56-45-1, L-Serine, reactions 61-90-5, L-Leucine, reactions 63-68-3, L-Methionine, reactions 63-91-2, L-Phenylalanine, reactions 70-47-3, L-Asparagine, reactions 74-79-3, L-**Arginine**, reactions 147-85-3, L-Proline, reactions
RL: RCT (Reactant); RACT (Reactant or reagent)
(triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root))

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> d total ibib abs

L6 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:120414 CAPLUS
DOCUMENT NUMBER: 144:184702
TITLE: Gene expression profiles for identifying patients at risk of developing encephalitis following immunotherapy for Alzheimer's disease
INVENTOR(S): O'Toole, Margot; Dorner, Andrew J.; Janszen, Derek B.; Slonim, Donna K.; Mounts, William M.; Reddy, Padmalatha S.; Hill, Andrew A.
PATENT ASSIGNEE(S): Wyeth, John, and Brother Ltd., USA
SOURCE: PCT Int. Appl., 298 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006014755	A2	20060209	WO 2005-US25771	20050720

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,

SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2004-589877P P 20040720
US 2005-672716P P 20050418

AB The present invention generally relates to a method for an improved treatment for Alzheimer's disease (AD) using immunotherapy, e.g., immunotherapy targeting β amyloid (Aβ), e.g. immunotherapy based on AN1792. By ANOVA and GeneCluster analyses of Affymetrix U133A GeneChip data, statistically significant assocns. were detected between the gene expression profiles of peripheral blood mononuclear cells of patients prior to immunization with AN1792 and the post-immunization odevelopment of encephalitis. In addition, statistically significant assocns. were found between the pre-immunization gene expression profil in PBMCs and post-immunization development of IgG response. The method allows for predicting an adverse clin. response, and therefore allows for an improved safety profile of AN1792. In another embodiment, the method allows for predicting a favorable clin. response, and therefore allows for an improved efficacy profile of AN1792. The methods of the present invention may be combined to predict a favorable clin. response and the lack of an adverse clin. response.

L6 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1148985 CAPLUS
DOCUMENT NUMBER: 144:210192
TITLE: Myeloperoxidase-Generated Oxidants Modulate Left Ventricular Remodeling but Not Infarct Size After Myocardial Infarction
AUTHOR(S): Vasilyev, Nikolay; Williams, Timothy; Brennan, Marie-Luise; Unzek, Samuel; Zhou, Xiaorong; Heinecke, Jay W.; Spitz, Douglas R.; Topol, Eric J.; Hazen, Stanley L.; Penn, Marc S.
CORPORATE SOURCE: Departments of Cell Biology, Cleveland Clinic Foundation, Cleveland, OH, USA
SOURCE: Circulation (2005), 112(18), 2812-2820
CODEN: CIRCAZ; ISSN: 0009-7322
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Background: Inflammation after myocardial infarction (MI) heralds worse left ventricular (LV) function and clin. outcomes. However, whether inflammation affects LV function by extending myonecrosis and/or altering LV remodeling remains unknown. We hypothesized that cytotoxic aldehydes generated during oxidative stress may adversely affect remodeling and infarct size. One theor. source of reactive aldehydes is oxidation of common α-amino acids by myeloperoxidase (MPO) released by leukocytes. However, a role for MPO in formation of aldehydes in vivo and the functional consequences of MPO-generated oxidants in **ischemia/reperfusion** models of MI have not been established. Methods and Results: In studies with cell types found in vascular tissue, MPO-oxidation products of **glycine** (formaldehyde) and threonine (acrolein) were the most cytotoxic. Mass spectrometry studies of myocardial tissue from murine models of acute MI (both chronic left anterior descending coronary artery ligation and **ischemia/reperfusion** injury) confirmed that MPO serves as a major enzymic source in the generation of these cytotoxic aldehydes. Interestingly, although MPO-null mice experienced 35.1% ($P<0.001$) less LV dilation and a 52.2% ($P<0.0001$) improvement in LV function compared with wild-type mice 24 days after **ischemia/reperfusion** injury, no difference in infarct size between wild-type and MPO-null mice was noted. Conclusions: The present data sep. inflammatory effects on infarct size and LV remodeling

and demonstrate that MPO-generated oxidants do not significantly affect tissue necrosis after MI but rather have a profound adverse effect on LV remodeling and function.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1109040 CAPLUS
DOCUMENT NUMBER: 144:147900
TITLE: Role of reactive oxygen species, nitric oxide and mitochondrial KATP channels in tumor necrosis factor (TNF)-alpha induced cardioprotection
AUTHOR(S): Fu, Chen; Xia, Qiang; Cao, Chunmei; Gao, Qin; Yao, Hui; Jin, Hongfeng
CORPORATE SOURCE: School of Medicine, Zhejiang University, Hangzhou, 310031, Peop. Rep. China
SOURCE: Zhongguo Yingyong Shenglixue Zazhi (2005), 21(1), 20-24
CODEN: ZYSZE2; ISSN: 1000-6834
PUBLISHER: Zhongguo Yingyong Shenglixue Zazhi Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The cardiac effect of tumor necrosis factor-α (TNF-α) in postischemic heart and the possible mechanism were explored. Langendorff perfused rat heart was used to evaluate the contractile properties of myocardium by intraventricular pressure measurement. The isolated rat heart underwent 20 min of global ischemia followed by 20 min of reperfusion. Level of lactate dehydrogenase (LDH) in the coronary effluent was measured to evaluate the cardiac injury. And the activity of manganese superoxide dismutase (Mn-SOD) in myocardial mitochondria was measured. Perfusion with TNF-α (104 U/L) attenuated the inhibitory effects induced by **ischemia/reperfusion** on left ventricular developed pressure (LVDP), left ventricular end-diastolic pressure (LVEDP), maximal rise/fall rate of left ventricular pressure (±dP/dt max) and rate pressure product (LVDP multiplied by heart rate, LVDP+HR). TNF-α significantly decreased the release of LDH ($P<0.05$) in the coronary effluent and increased the activity of Mn-SOD in the myocardial mitochondria ($P<0.01$). Antioxidant N-(2-mercaptopropionyl) **glycine** (2-MPG, 0.3 mmol/L), nitric oxide synthase (NOS) inhibitor NG-nitro-L-**arginine** Me ester (L-NAME, 0.5 mmol/L) or mitochondrial selective KATP channel inhibitor 5-hydroxydecanoate (5-HD, 100 μmol/L) attenuated the increase in LVDP, ±dP/dtmax and LVDP+HR, and decrease in LVEDP induced by TNF-α in **ischemia/reperfusion** heart, resp. And the effects of TNF-α in reducing the levels of LDH and increasing the Mn-SOD activity were also attenuated by 2-MPG, L-NAME or 5-HD, resp. TNF-α pretreatment attenuates the myocardial injury induced by **ischemia /reperfusion**, which coincides with the increasing of myocardial Mn-SOD activity. Reactive oxygen species, nitric oxide and mitochondrial KATP channels are involved in the cardioprotection induced by TNF-α.

L6 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:337852 CAPLUS
DOCUMENT NUMBER: 143:4351
TITLE: mitoKATP channel activation in the postanoxic developing heart protects E-C coupling via NO-, ROS-, and PKC-dependent pathways
AUTHOR(S): Sarre, Alexandre; Lange, Norbert; Kucera, Pavel; Raddatz, Eric
CORPORATE SOURCE: Department of Physiology, Faculty of Biology and Medicine, University of Lausanne, Lausanne, Switz.
SOURCE: American Journal of Physiology (2005), 288(4, Pt. 2), H1611-H1619
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Whereas previous studies have shown that opening of the mitochondrial ATP-sensitive K+ (mitoKATP) channel protects the adult heart against **ischemia-reperfusion** injury, it remains to be established whether this mechanism also operates in the developing heart. Isolated spontaneously beating hearts from 4-day-old chick embryos were subjected to 30 min of anoxia followed by 60 min of reoxygenation. The chrono-, dromo-, and inotropic disturbances, as well as alterations of the electromech. delay (EMD), reflecting excitation-contraction (E-C) coupling, were investigated. Production of reactive oxygen species (ROS) in the ventricle was determined using the intracellular fluorescent probe 2',7'-dichlorofluorescin (DCFH). Effects of the specific mitoKATP channel opener diazoxide (Diazo, 50 μM) or the blocker 5-hydroxydecanoate (5-HD, 500 μM), the nitric oxide synthase (NOS) inhibitor NG-nitro-L-**arginine** Me ester (L-NAME, 50 μM), the antioxidant N-(2-mercaptopropionyl) **glycine** (MPG, 1 mM), and the PKC inhibitor chelerythrine (Chel, 5 μM) on oxidative stress and postanoxic functional recovery were determined Under normoxia, the baseline parameters were not altered by any of these pharmacol. agents, alone or in combination. During the first 20 min of postanoxic reoxygenation, Diazo doubled the peak of ROS production and, interestingly, accelerated recovery of ventricular EMD and the PR interval. Diazo-induced ROS production was suppressed by 5-HD, MPG, or L-NAME, but not by Chel. Protection of ventricular EMD by Diazo was abolished by 5-HD, MPG, L-NAME, or Chel, whereas protection of the PR interval was abolished by L-NAME exclusively. Thus pharmacol. opening of the mitoKATP channel selectively improves postanoxic recovery of cell-to-cell communication and ventricular E-C coupling. Although the NO-, ROS-, and PKC-dependent pathways also seem to be involved in this cardioprotection, their interrelation in the developing heart can differ markedly from that in the adult myocardium.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:307068 CAPLUS
DOCUMENT NUMBER: 143:151083
TITLE: A Study of the Metabolites of **Ischemia-Reperfusion** Injury and Selected Amino Acids in the Liver Using Microdialysis during Transplantation
AUTHOR(S): Silva, Michael A.; Richards, Douglas A.; Bramhall, Simon R.; Adams, David H.; Mirza, Darius F.; Murphy, Nick
CORPORATE SOURCE: Liver Unit, University Hospital Birmingham NHS Trust, Birmingham, UK
SOURCE: Transplantation (2005), 79(7), 828-835
CODEN: TRPLAU; ISSN: 0041-1337
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Background: Preservation and **ischemia-reperfusion** injury still impact the outcome of orthotopic liver transplantation. The authors used microdialysis with a view to monitoring its effect on graft function. Methods: A microdialysis catheter was inserted into the graft immediately after reperfusion and perfused with an isotonic solution for 48 h. Metabolites of the **ischemia-reperfusion** injury and selected amino acids were studied. There were 18 patients, with a median age of 52 years (range, 38-62 years), 8 of whom were men. Lactate, pyruvate, glycerol, and glucose levels were measured. In addition, alanine, **arginine**, citrulline, γ-aminobutyric acid (GABA), glutamate, glutamine, **glycine**, and taurine were determined Results: All grafts functioned well. High lactate, pyruvate, and glycerol levels were observed in the immediate postoperative period. These showed a significant rapid decrease and stabilized to baseline levels. Alanine, glutamate, GABA, and taurine levels declined significantly to baseline values.

Arginine levels were low immediately postreperfusion and then increased, reaching significantly higher values beyond 19 h. Conclusions: These data may represent "normal" changes seen in the immediate posttransplant period because all grafts functioned well. Two important metabolic fates of **arginine** in the liver are in the detoxification of ammonia by means of the urea cycle, and in the synthesis of nitric oxide (NO). Low extracellular **arginine** may reflect influx of the amino acid into hepatocytes, resulting in formation of NO in the presence of inducible NO synthase or conversion to ornithine in the presence of arginase in the urea cycle. As the organ stabilizes, restriction of **arginine** uptake may give rise to the observed increase in extracellular **arginine.**

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:494285 CAPLUS
DOCUMENT NUMBER: 141:421923
TITLE: Alleviating **ischemia-reperfusion** injury in small bowel
AUTHOR(S): Salehi, Payam; Madsen, Karen; Zhu, Jay; Castillo, Erika; Avila, Jose; Lakey, Jonathan R. T.; Churchill, Thomas A.
CORPORATE SOURCE: Surgical-Medical Research Institute, University of Alberta, Edmonton, AB, T6G 2N8, Can.
SOURCE: American Journal of Transplantation (2004), 4(5), 728-737
CODEN: AJTMBR; ISSN: 1600-6135
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An amino acid-based solution has been recently developed and has demonstrated significant protective effects during cold storage of small bowel (SB). This study was designed to examine the role of this novel solution in ameliorating intestinal injury in an in vivo model of **ischemia-reperfusion** (IR). The impact of luminal treatment with an amino acid-based (AA) solution was assessed throughout reperfusion after 60-min warm ischemia (WI) in rodent SB. Energetics (ATP and total adenylates) remained significantly elevated throughout 60-min reperfusion in AA-treated tissue compared with untreated controls. Increases in end-products (ammonia and alanine) and increases in alanine aminotransferase and glutaminase activity implicated greater amino acid metabolism in AA-treated tissues. After reperfusion, malondialdehyde levels were similar between all groups. Glutathione levels were consistently elevated in AA-treated tissues and by 60 min reperfusion values were sixfold greater than control. AA-mediated protection during IR resulted in reduced neutrophil infiltration suggesting a weaker inflammatory response. Barrier function and electrophysiol. parameters exhibited a clear pattern of mucosal preservation in AA-treated tissues; histol. supported these findings. This study raises the possibility of a role for a luminal nutrient-rich solution during ischemic storage of small bowel in the clinic.

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:613029 CAPLUS
DOCUMENT NUMBER: 140:300402
TITLE: Triterpenoid constituents with nitric oxide production inhibitory activity from several fragrance herbal medicines (myrrh, olibanum, and saussurea root)
AUTHOR(S): Morikawa, Toshio; Matsuda, Hisashi; Oominami, Hideo; Kageura, Tadashi; Toguchida, Iwao; Yoshikawa, Masayuki
CORPORATE SOURCE: Kyoto Pharmaceutical University, Japan
SOURCE: Tennen Yuki Kagobutsu Toronkai Koen Yoshishu (2001),

43rd, 485-490
CODEN: TYKYDS
PUBLISHER: Nippon Kagakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB Nitric oxide (NO), an inorg. free radical, has been implicated in physiol. and pathol. process such as vasodilation, non-specific host defense, **ischemia reperfusion** injury and chronic inflammation. NO is produced by the oxidation of L-**arginine** by a NO synthase (NOS). In the family of NOS, inducible NOS in particular is involved in a pathol. aspect with overprodn. of NO, and can be expressed in response to pro-inflammatory, agents such as interleukin-1β, tumor necrosis factor-α, and lipopolysaccharide (LPS) in various cells including macrophages, endothelial cells, and smooth muscle cells. In the course of the authors' characterization studies on bioactive constituents of natural medicines, the authors found that the methanolic or 80% aqueous acetone extract of several fragrance herbal medicines such as Myrrh (the resin of Baltamodendron mukul HOOK.), Olibanum (the resin of Boswellia carterri BIRDW.), and Saussurea root (the root of Saussurea lappa CLARKE) showed NO production inhibitory activity in LPS-activated macrophages. Through bioassay-guided separation, four new bicyclic triterpene constituents, myrrhanols A and B and myrrhanones A and B were isolated from the methanolic extract of Indian Myrrh together with fourteen known compds. including a progestational hormone, progesterone. On the similar procedure, the 80% aqueous acetone extract of Egyptian Olibanum was purified by various chromatogs. to furnish thirteen new terpene constituents named olibanumols A-M together with twenty-nine known compds. such as epilupeol. The stereostructures of their new constituents were elucidated on the basis of chemical and physicochem. evidence. In addition, saussureamines A-E, five new amino acid-sesquiterpene conjugates, were isolated from the methanolic extract of Chinese Saussurea root together with costunolide and dehydrocosms lactone etc. The absolute stereostrocutres of saussureamines A-E were determined on the basis of synthetic evidence. Thus, saussureamins A-E and the related amino acid-sesquiterpene conjugates were synthesized using Michael type addition reaction of amino acid to the α-methylene-γ-lactone moiety of sesquiterpenes. Finally, the isolated constituents such as bicyclic triterpenoids (myrrhanols A and B, myrrhanones A and B) from Myrrh, lupane- and ursane-type triterpenoids and dammarane-type nortriterpenoid (hip-20(30)-ene-3,29-diol, urs-12-ene35,lla-diol, 30-hydroxymansumbin-13(17)-ene-16-one) from Olibanum, and amino acid-sesquiterpene conjugates (saussureamines A and B) from Saussurea root were found to strongly inhibit the NO production Saussureamines A and B inhibited iNOS induction in accordance with induction of heat shock protein 72 (HSP 72). These results suggested that, as one of their mechanisms of action, amino acid-sesquiterpene conjugates induced HSP 72 thereby preventing nuclear factor-.vkappa.B activation followed by iNOS induction.

L6 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:536296 CAPLUS
DOCUMENT NUMBER: 137:367870
TITLE: Effect of Nitric Oxide on the Contractile Function of Rat Reperfused Skeletal Muscle
AUTHOR(S): Ikebe, Kenshiro; Kato, Teiji; Yamaga, Makio; Tsuchida, Toru; Irie, Hiroki; Oniki, Yasunari; Takagi, Katsumasa
CORPORATE SOURCE: Department of Orthopedic Surgery, Kumamoto University School of Medicine, Kumamoto, Japan
SOURCE: Journal of Surgical Research (2002), 106(1), 82-85
CODEN: JSGRA2; ISSN: 0022-4804
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The involvement of nitric oxide (NO) in **ischemia-reperfusion** injury remains controversial and has been reported to be both beneficial and deleterious. The purpose of this study was to

examine the contribution of NO and superoxide to skeletal muscle function using an ischemic revascularized hind limb model in rats. Warm ischemia produced by vascular pedicle clamping was sustained for 3 h. The animals were divided into four groups according to the solution administrated: (1) saline, (2) N-methyl-L-**arginine** acetate (L-NMMA), (3) L-NMMA + N-(N-L-g-glutamyl-S-nitroso-l-cysteinyl)**glycine** (S-nitrosoglutathione), or (4) superoxide dismutase (SOD). Saline, L-NMMA, or L-NMMA + S-nitrosoglutathione was infused for the first 2 h of reperfusion. The SOD was administered as an i.v. bolus 5 min before the onset of reperfusion. Postischemic blood flow was measured by a Doppler flow meter. Muscle contractile function was determined after 24 h of reperfusion.Results. Postischemic blood flow was significantly decreased by the L-NMMA infusion compared with that in the saline-treated group. No significant difference in postischemic blood flow was noted in the saline-, L-NMMA + S-nitrosoglutathione-, and SOD-treated groups. Contractile function of the gastrocnemius muscle in the L-NMMA-and SOD-treated groups, but not in the L-NMMA + S-nitrosoglutathione group, was significantly better than that in the saline-treated group. Limiting postischemic blood flow and SOD infusion are both beneficial in decreasing the **ischemia-reperfusion** injury of skeletal muscle. S-Nitrosoglutathione infusion following suppression of endogenous NO production does not reduce **ischemia-reperfusion** injury.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1999:537306 CAPLUS

DOCUMENT NUMBER: 132:34225

TITLE: Differences in osteopontin up-regulation between proximal and distal tubules after renal **ischemia/reperfusion**

AUTHOR(S): Persy, Veerle P.; Verstrepen, Walter A.; Ysebaert, Dirk K.; De Greef, Kathleen E.; De Broe, Marc E.

CORPORATE SOURCE: Departments of Nephrology and Experimental Surgery, University of Antwerp, Antwerp, Belg.

SOURCE: Kidney International (1999), 56(2), 601-611 CODEN: KDYIA5; ISSN: 0085-2538

PUBLISHER: Blackwell Science, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Osteopontin (OPN) is a highly acidic phosphoprotein containing an **arginine-glycine**-aspartic acid (RGD) cell adhesion motif. High OPN expression has been found in tissues with high cell turnover, and OPN up-regulation has been demonstrated in several models of renal injury, suggesting a possible role in tissue remodeling and repair. However, its exact function in the kidney remains unknown. In this study, the possible contribution of OPN to regeneration and repair in the kidney was explored by studying the time course and subcellular localization of OPN up-regulation after renal **ischemia/reperfusion** injury in different nephron segments and by investigating its relationship with tubular morphol. Rats that underwent 60 min of left renal ischemia and a right nephrectomy sacrificed at 10 different time points (from 1 h to 10 days after reperfusion) were compared with uninephrectomized rats at each time point. In renal tissue sections immunostained for OPN, proximal (PTs) and distal tubules (DTs) in both the renal cortex and outer stripe of the outer medulla (OSOM) were scored for the degree of OPN expression and tubular morphol. Kidneys of uninephrectomized rats showed no injury, and the localization and intensity of their OPN expression remained unaltered compared with normal rats. After **ischemia/reperfusion**, morphol. damage was most severe in PTs of the OSOM, but all examined nephron segments showed a significant increase in OPN expression. The time course of OPN up-regulation was different in PTs and DTs. DTs in both cortex and OSOM rapidly increased their OPN expression, with a maximum at 24 h after reperfusion followed by a slow decrease. In contrast, PTs showed a delayed increase in OPN staining, with a maximum after

five to seven days, higher in the OSOM than in the cortex. In OSOM PTs, OPN expression was predominantly associated with morphol. regeneration, whereas DTs showed a substantial OPN up-regulation without major morphol. damage. PTs and DTs displayed a different subcellular OPN staining pattern: OPN staining in DTs was located to the apical side of the cell; PTs, however, presented a vesicular, perinuclear staining pattern. Our study found a different pattern of OPN up-regulation after renal **ischemia/reperfusion** in PTs vs. DTs, both with regard to time course and subcellular localization. DTs show an early and persistent increase in OPN staining in the absence of major morphol. injury, whereas OPN staining in PTs is delayed and is mostly associated with morphol. regeneration. PTs show a vesicular, perinuclear OPN staining pattern, whereas DTs show OPN staining at the apical cell side.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1997:336210 CAPLUS

DOCUMENT NUMBER: 127:711

TITLE: Modulation of ischemic excitatory neurotransmitter and γ-aminobutyric acid release during global temporary cerebral ischemia by local nitric oxide synthase inhibition

AUTHOR(S): Kahn, Ronald A.; Panah, Michael; Kiffel, Steven; Weinberger, Jesse

CORPORATE SOURCE: Departments of Anestesiology, Mount Sinai Medical Center, New York, NY, USA

SOURCE: Anesthesia & Analgesia (Baltimore) (1997), 84(5), 1004-1010
CODEN: AACRAT; ISSN: 0003-2999

PUBLISHER: Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Systemic nitric oxide synthase inhibition (NOSI) decreases cerebral blood flow, which may worsen ischemic insults. To examine the local effects of NOSI without this confounding effect, we examined the role of a locally administered NOSI, NG-nitro-L-**arginine**-methyl-ester (L-NAME), on neurotransmitter recovery during cerebral ischemia. Rats were assigned to one of three groups: locally administered L-NAME via a striatal microdialysis probe (n=11). Temporary global forebrain ischemia was induced for 15 min, followed by 60 min of reperfusion. L-NAME resulted in decreases of basal aspartate (ASP; 74% of basal) and glutamate (GLU; 60% of basal) recovery. While systemic L-NAME caused significant increases in ischemic ASP and GLU recovery (by 224% and 110%, resp., compared with ischemic controls), local NOSI administration resulted in a significant attenuation of peak ASP, GLU, **glycine**, and γ-aminobutyric acid recovery (43%, 38% 53%, and 72%, resp., compared with ischemic controls). We conclude that local NOSI attenuated ischemic neurotransmitter recovery during **ischemia-reperfusion.** Our results emphasize the importance of the systemic effects of NOSI and suggest both deleterious and beneficial effects of NOSI during **ischemia/reperfusion.**

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1993:252488 CAPLUS

DOCUMENT NUMBER: 118:252488

TITLE: Arterial endothelial barrier dysfunction: Actions of homocysteine and the hypoxanthine-xanthine oxidase free radical generating system

AUTHOR(S): Berman, Rodney S.; Martin, William

CORPORATE SOURCE: Dep. Pharmacol., Univ. Glasgow, Glasgow, G12 8QQ, UK

SOURCE: British Journal of Pharmacology (1993), 108(4), 920-6
CODEN: BJPCBM; ISSN: 0007-1188

DOCUMENT TYPE: Journal
LANGUAGE: English

AB Endothelial barrier function was assessed by use of an in vitro model in which transfer of trypan blue-labeled albumin was measured across monolayers of bovine aortic endothelial cells grown on polycarbonate membranes. Addition of either hypoxanthine (0.2 mM) or xanthine oxidase (20 mu mL-1) alone during a 90 min incubation did not affect albumin transfer across endothelial cell monolayers, but a combination of both increased transfer. The increase in albumin transfer induced by hypoxanthine and xanthine oxidase was abolished by catalase (3 u mL-1), reduced by allopurinol (4 mM), but unaffected by superoxide dismutase (6000 u mL-1), the hydroxyl radical scavengers, mannitol (15 mM), dimethylthiourea (10 mM) and N-(2-mercaptopropionyl)-**glycine** (1 mM), the iron chelator, deferoxamine (0.5 mM), ferric chloride (50 μM), and inhibitor of nitric oxide synthase, NG-nitro-L-**arginine** (30 μM), or the antioxidant, dithiothreitol (3 mM). Hydrogen peroxide (0.1-30 mM) itself increased albumin transfer across endothelial cell monolayers, exhibiting a biphasic concentration-response curve. The increase induced by 0.1 mM hydrogen peroxide was abolished in the presence of 0.3 m mL-1 catalase whilst that induced by 10 mM hydrogen peroxide was abolished by 3000 u mL-1 catalase. Homocysteine (0.5-1.5 mM) did not affect albumin transfer across endothelial monolayers when added alone, but when added in combination with copper sulfate (50 μM), which catalyzes its oxidation, a significant increase in albumin transfer was observed The increase in albumin transfer induced by the combination of homocysteine (1.5 mM) and copper sulfate was abolished by catalase (1 u mL-1), but was unaffected by superoxide dismutase (6000 u mL-1), mannitol (15 mM), dimethylthiourea (1 mM) or deferoxamine (0.5 mM). The data suggest that the endothelial barrier dysfunction induced by the combination of hypoxanthine and xanthine oxidase is mediated solely by the action of hydrogen peroxide and not by superoxide anion, hydroxyl radical, peroxynitrite anion or hypochlorous acid. The copper-catalyzed oxidation of homocysteine also induced endothelial barrier dysfunction through the generation of hydrogen peroxide. These findings may have relevance to the endothelial barrier dysfunction associated with **ischemia-reperfusion** injury and the atherogenic actions of homocysteine.

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=> s (green tea extract### or catechin or epicatechin or epicgallocatechin) and
(nitric oxide donor)
        250222 GREEN
          2406 GREENS
        251551 GREEN
                  (GREEN OR GREENS)
         33536 TEA
          1699 TEAS
         33862 TEA
                  (TEA OR TEAS)
        250253 EXTRACT###
        304553 EXT
        223333 EXTS
        470842 EXT
                  (EXT OR EXTS)
        352214 EXTD
             7 EXTDS
        352216 EXTD
                  (EXTD OR EXTDS)
         54840 EXTG
             1 EXTGS
         54841 EXTG
                  (EXTG OR EXTGS)
        391734 EXTN
         14061 EXTNS
        397260 EXTN
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              (EXTN OR EXTNS)
      1065363 EXTRACT###
              (EXTRACT### OR EXT OR EXTD OR EXTG OR EXTN)
         1037 GREEN TEA EXTRACT###
              (GREEN(W)TEA(W)EXTRACT###)
         8302 CATECHIN
         3033 CATECHINS
         9363 CATECHIN
              (CATECHIN OR CATECHINS)
         4662 EPICATECHIN
           61 EPICATECHINS
         4676 EPICATECHIN
              (EPICATECHIN OR EPICATECHINS)
            0 EPICGALLOCATECHIN
       167016 NITRIC
            3 NITRICS
       167019 NITRIC
              (NITRIC OR NITRICS)
      1637509 OXIDE
       338424 OXIDES
      1733818 OXIDE
              (OXIDE OR OXIDES)
       150066 DONOR
        71942 DONORS
       192456 DONOR
              (DONOR OR DONORS)
         2279 NITRIC OXIDE DONOR
              (NITRIC(W)OXIDE(W)DONOR)
L7          4 (GREEN TEA EXTRACT### OR CATECHIN OR EPICATECHIN OR EPICGALLOCAT
              ECHIN) AND (NITRIC OXIDE DONOR)

=> d total ibib abs

L7   ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2004:513526  CAPLUS
DOCUMENT NUMBER:          141:47384
TITLE:                    Gastrointestinally deliverable formulation containing
                          green tea extract and a
                          nitric oxide donor for the
                          reduction of postoperative complications
INVENTOR(S):              Schneider, Heinz
PATENT ASSIGNEE(S):       Fresenius Kabi Deutschland GmbH, Germany
SOURCE:                   PCT Int. Appl., 21 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 German
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     --------------       ----   --------    --------------------    --------
     WO 2004052352         A1    20040624    WO 2003-EP12675         20031113
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO,
             NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ,
             TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
             TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     DE 10257360           A1    20040708    DE 2002-10257360        20021209
     CA 2499006            AA    20040624    CA 2003-2499006         20031113
     AU 2003288047         A1    20040630    AU 2003-288047          20031113
```

BR 2003015075	A	20050816	BR 2003-15075	20031113
EP 1572175	A1	20050914	EP 2003-779907	20031113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
NO 2005002978	A	20050617	NO 2005-2978	20050617
PRIORITY APPLN. INFO.:			DE 2002-10257360	A 20021209
			WO 2003-EP12675	W 20031113

AB The invention discloses a formulation, which can be administered gastrointestinally, containing **green tea extract** and at least one nitric oxide (NO) donor (or precursor thereof) which is a substrate of NO synthetase. The formulation is administered prior to surgical interventions, to eliminate or reduce the risk of postoperative complications.

L7 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER:	1999:310369 CAPLUS
DOCUMENT NUMBER:	131:98618
TITLE:	In vitro cytotoxicity of the **nitric oxide donor**, S-nitroso-N-acetyl-penicillamine, towards cells from human oral tissue
AUTHOR(S):	Babich, Harvey; Zuckerbraun, Harriet L.; Hirsch, Shoshana T.; Blau, Lea
CORPORATE SOURCE:	Department of Biology, Stern College for Women, Yeshiva University, New York, NY, 10016, USA
SOURCE:	Pharmacology & Toxicology (Copenhagen) (1999), 84(5), 218-225 CODEN: PHTOEH; ISSN: 0901-9928
PUBLISHER:	Munksgaard International Publishers Ltd.
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB The cytotoxicity of the **nitric oxide donor**, S-nitroso-N-acetyl-penicillamine (SNAP), towards cultured human cells from oral tissue was evaluated. The toxicity of SNAP to Smulow-Glickman gingival epithelial cells was correlated with the liberation of nitric oxide, as N-acetyl-DL-penicillamine, the SNAP metabolites, N-acetyl-DL-penicillamine disulfide and nitrite, and preincubated (denitrosylated) SNAP did not affect viability. Comparing equimolar concns. of various **nitric oxide donors**, cytotoxicity appeared to be inversely related to the relative stability (i.e., half-life) of the test compound; the sequence of cytotoxicity for a 4 h exposure was S-nitrosoglutathione >> spermine NONOate > SNAP > DPTA NONOate >> DETA NONOate. Intracellular reduced glutathione (GSH) was lowered in S-G cells exposed to SNAP. Pretreatment of the cells with the GSH depleter, 1,3-bis-(chloroethyl)-1-nitrosourea (BCNU), enhanced the toxicity of SNAP. Similar findings of enhanced sensitivity to SNAP were noted with gingival fibroblasts and periodontal ligament cells pretreated with BCNU. The toxicity of SNAP towards the gingival epithelial cells was decreased by cotreatment with the antioxidants, N-acetyl-L-cysteine, L-ascorbic acid, and (+)-**catechin.** Cells exposed to SNAP exhibited nuclear aberrations, including multilobed nuclei and multinucleation. SNAP-induced cell death was apparently by apoptosis, as noted by fluorescence microscopy and DNA agarose gel electrophoresis.

REFERENCE COUNT:	48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER:	1999:246034 CAPLUS
DOCUMENT NUMBER:	131:28775
TITLE:	Aortic endothelial cells damaged by a **nitric oxide donor** and protected by flavonoids
AUTHOR(S):	Law, Ada; Wu, Jun; Zeng, Ling-Hua; Wu, Tai-Wing
CORPORATE SOURCE:	Department of Clinical Biochemistry, University of Toronto, Toronto, M5T 2S8, Can.
SOURCE:	Life Sciences (1999), 64(19), PL199-PL204

CODEN: LIFSAK; ISSN: 0024-3205
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Cultured porcine aortic endothelial cells (PAEC) were exposed to four concns. (0.00 mM - 5.00 mM) of 3-morpholinosydnonimine hydrochloride (SIN-1, a **nitric oxide donor**). SIN-1 demonstrated a dose dependent cytotoxicity against PAEC as indicated by the thiobarbituric acid (TBA) assay. Morphol. and biochem., the presence of selected flavonoids (morin, quercetin, or **catechin**) was shown to protect the PAEC from SIN-1 toxicity. Protection levels determined from the TBA assay were significant ($p<0.05$) for all flavonoids, with morin at 72±8%. Quercetin and **catechin** had comparable protective activities of 54±6% and 43±3%, resp. This study supports the contention that SIN-1 is cytotoxic to PAEC and that antioxidants such as flavonoids may attenuate such toxicity.
REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:92760 CAPLUS
TITLE: In vitro cytotoxicity of the **nitric oxide donor**, S-nitroso-N-acetyl-penicillamine, toward cells from human oral tissue
AUTHOR(S): Blau, L.; Babich, H.; Zuckerbraun, H. L.; Hirsch, S. T.
CORPORATE SOURCE: Stern College for Women, Yeshiva University, New York, NY, 10016, USA
SOURCE: Book of Abstracts, 217th ACS National Meeting, Anaheim, Calif., March 21-25 (1999), MEDI-246. American Chemical Society: Washington, D. C.
CODEN: 67GHA6
DOCUMENT TYPE: Conference; Meeting Abstract
LANGUAGE: English
AB The cytotoxicity of the **nitric oxide donor**, S-nitroso-N-acetyl-penicillamine (SNAP), towards cultured human cells from oral tissue was evaluated. The toxicity of SNAP to Smulow-Glickman gingival epithelial cells was correlated with its release of NO. The cytotoxicity of a number of NO donors appeared to be inversely related to their relative rates of NO release as expressed in half-lives of the reaction. It was found that the sequence of cytotoxicity for a 4-h exposure was S-nitrosoglutathione>>spermine NONOate>SNAP>DPTA NONOate>> DETA NONOate. Intracellular reduced glutathione (GSH) was lowered in Smulow-Glickman cells exposed to SNAP. Pretreatment of the cells with the GSH depleter, 1,3-bis-(chloroethyl)-1-nitrosourea (BCNU), enhanced the toxicity of SNAP. Similar findings were observed in gingival fibroblasts and periodontal ligament cells. Treatment of the gingival epithelial cells with antioxidants, N-acetyl-L-cysteine, L-ascorbic acid, or (+)-**catechin**, in presence of SNAP, reduced SNAP toxicity. Cells exposed to SNAP exhibited nuclear aberrations, including multilobed nuclei and multinucleation. SNAP-induced cell death was apparently by apoptosis, as noted by fluorescence microscopy and DNA agarose gel electrophoresis.

=> s (green tea extract### or catechin or epicatechin or epicgallocatechin) and (ischemia reperfusion or oxygen reperfusion)
250222 GREEN
2406 GREENS
251551 GREEN
(GREEN OR GREENS)
33536 TEA
1699 TEAS
33862 TEA
(TEA OR TEAS)
250253 EXTRACT###

```
        304553 EXT
        223333 EXTS
        470842 EXT
                 (EXT OR EXTS)
        352214 EXTD
             7 EXTDS
        352216 EXTD
                 (EXTD OR EXTDS)
         54840 EXTG
             1 EXTGS
         54841 EXTG
                 (EXTG OR EXTGS)
        391734 EXTN
         14061 EXTNS
        397260 EXTN
                 (EXTN OR EXTNS)
       1065363 EXTRACT###
                 (EXTRACT### OR EXT OR EXTD OR EXTG OR EXTN)
          1037 GREEN TEA EXTRACT###
                 (GREEN(W)TEA(W)EXTRACT###)
          8302 CATECHIN
          3033 CATECHINS
          9363 CATECHIN
                 (CATECHIN OR CATECHINS)
          4662 EPICATECHIN
            61 EPICATECHINS
          4676 EPICATECHIN
                 (EPICATECHIN OR EPICATECHINS)
             0 EPICGALLOCATECHIN
         66759 ISCHEMIA
            71 ISCHEMIAS
         66774 ISCHEMIA
                 (ISCHEMIA OR ISCHEMIAS)
         28274 REPERFUSION
            52 REPERFUSIONS
         28283 REPERFUSION
                 (REPERFUSION OR REPERFUSIONS)
         14410 ISCHEMIA REPERFUSION
                 (ISCHEMIA(W)REPERFUSION)
        713571 OXYGEN
          6785 OXYGENS
        718307 OXYGEN
                 (OXYGEN OR OXYGENS)
         28274 REPERFUSION
            52 REPERFUSIONS
         28283 REPERFUSION
                 (REPERFUSION OR REPERFUSIONS)
            25 OXYGEN REPERFUSION
                 (OXYGEN(W)REPERFUSION)
L8           32 (GREEN TEA EXTRACT### OR CATECHIN OR EPICATECHIN OR EPICGALLOCAT
                ECHIN) AND (ISCHEMIA REPERFUSION OR OXYGEN REPERFUSION)

=> dup rem
ENTER L# LIST OR (END):l8
PROCESSING COMPLETED FOR L8
L9            32 DUP REM L8 (0 DUPLICATES REMOVED)

=> d scan

L9    32 ANSWERS   CAPLUS  COPYRIGHT 2006 ACS on STN
CC    1-12 (Pharmacology)
TI    Catecholic iron complexes as cytoprotective superoxide scavengers against
      hypoxia/reoxygenation injury in isolated hepatocytes
ST    catechol iron complex hepatoprotectant reperfusion injury; antioxidant
      superoxide scavenger catechol iron complex
```

IT Antioxidants
Hypoxia, animal
Radical scavengers
(catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)
IT Flavanols
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)
IT Reactive oxygen species
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)
IT Cytoprotective agents
(hepatoprotectants; catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)
IT Reperfusion
(injury; catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)
IT 98-29-3D, 4-tert-Butylcatechol, iron complexes 99-50-3D, Protocatechuic acid, iron complexes 117-39-5D, Quercetin, iron complexes 120-80-9D, Catechol, iron complexes 149-45-1D, Tiron, iron complexes 154-23-4D, **Catechin**, iron complexes 331-39-5D, Caffeic acid, iron complexes 452-86-8D, 4-Methylcatechol, iron complexes 488-17-5D, 3-Methylcatechol, iron complexes 490-46-0D, **Epicatechin**, iron complexes 1198-55-6D, Tetrachlorocatechol, iron complexes 3316-09-4D, 4-Nitrocatechol, iron complexes 7439-89-6D, Iron, complexes with catechols, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)
IT 11062-77-4, Superoxide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)
IT 9054-89-1, Superoxide dismutase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(mimics; catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L9 32 ANSWERS CAPLUS COPYRIGHT 2006 ACS on STN
CC 18-7 (Animal Nutrition)
Section cross-reference(s): 14, 17
TI Protective effect of **green tea extract** on **ischemia/reperfusion**-induced brain injury in Mongolian gerbils
ST **green tea ext** oxidative damage brain injury
IT Tea products
(beverages, green; **green tea extract** effect on **ischemia/reperfusion**-induced brain injury in Mongolian gerbils)
IT Brain
(cerebral cortex; **green tea extract** effect on **ischemia/reperfusion**-induced brain injury in Mongolian gerbils)
IT Brain

(corpus striatum; **green tea extract** effect
on **ischemia/reperfusion**-induced brain injury in
Mongolian gerbils)
IT DNA
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological
study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC
(Process)
(damage, oxidative; **green tea extract** effect
on **ischemia/reperfusion**-induced brain injury in
Mongolian gerbils)
IT Brain, disease
Reperfusion
(injury; **green tea extract** effect on
ischemia/reperfusion-induced brain injury in
Mongolian gerbils)
IT Behavior
(locomotor; **green tea extract** effect on
ischemia/reperfusion-induced brain injury in
Mongolian gerbils)
IT Cell death
(neuron; **green tea extract** effect on
ischemia/reperfusion-induced brain injury in
Mongolian gerbils)
IT Lipids, biological studies
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological
study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC
(Process)
(peroxidn.; **green tea extract** effect on
ischemia/reperfusion-induced brain injury in
Mongolian gerbils)
IT 7722-84-1, Hydrogen peroxide, biological studies
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological
study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC
(Process)
(**green tea extract** effect on
ischemia/reperfusion-induced brain injury in
Mongolian gerbils)

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> d title
'TITLE' IS NOT A VALID FORMAT FOR FILE 'CAPLUS'

The following are valid formats:

ABS --------- GI and AB
ALL --------- BIB, AB, IND, RE
APPS -------- AI, PRAI
BIB --------- AN, plus Bibliographic Data and PI table (default)
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DMAX -------- MAX, delimited for post-processing
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FBIB -------- AN, BIB, plus Patent FAM
IND --------- Indexing data
IPC --------- International Patent Classifications
MAX --------- ALL, plus Patent FAM, RE
PATS -------- PI, SO
SAM --------- CC, SX, TI, ST, IT
SCAN -------- CC, SX, TI, ST, IT (random display, no answer numbers;
SCAN must be entered on the same line as the DISPLAY,
e.g., D SCAN or DISPLAY SCAN)
STD --------- BIB, CLASS

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IABS -------- ABS, indented with text labels
IALL -------- ALL, indented with text labels
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OIBIB ------- OBIB, indented with text labels

SBIB -------- BIB, no citations
SIBIB ------- IBIB, no citations

HIT --------- Fields containing hit terms
HITIND ------ IC, ICA, ICI, NCL, CC and index field (ST and IT)
              containing hit terms
HITRN ------- HIT RN and its text modification
HITSTR ------ HIT RN, its text modification, its CA index name, and
              its structure diagram
HITSEQ ------ HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
KWIC -------- Hit term plus 20 words on either side
OCC --------- Number of occurrence of hit term and field in which it occurs

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All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR,
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to view a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):d ibib
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ALL --------- BIB, AB, IND, RE
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CBIB -------- AN, plus Compressed Bibliographic Data
CLASS ------- IPC, NCL, ECLA, FTERM
DALL -------- ALL, delimited (end of each field identified)
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FAM --------- AN, PI and PRAI in table, plus Patent Family data
FBIB -------- AN, BIB, plus Patent FAM
IND --------- Indexing data
IPC --------- International Patent Classifications
MAX --------- ALL, plus Patent FAM, RE
PATS -------- PI, SO
SAM --------- CC, SX, TI, ST, IT
SCAN -------- CC, SX, TI, ST, IT (random display, no answer numbers;
              SCAN must be entered on the same line as the DISPLAY,
              e.g., D SCAN or DISPLAY SCAN)
STD --------- BIB, CLASS

IABS -------- ABS, indented with text labels
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IALL -------- ALL, indented with text labels
IBIB -------- BIB, indented with text labels
IMAX -------- MAX, indented with text labels
ISTD -------- STD, indented with text labels

OBIB -------- AN, plus Bibliographic Data (original)
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SBIB -------- BIB, no citations
SIBIB ------- IBIB, no citations

HIT --------- Fields containing hit terms
HITIND ------ IC, ICA, ICI, NCL, CC and index field (ST and IT)
              containing hit terms
HITRN ------- HIT RN and its text modification
HITSTR ------ HIT RN, its text modification, its CA index name, and
              its structure diagram
HITSEQ ------ HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
KWIC -------- Hit term plus 20 words on either side
OCC --------- Number of occurrence of hit term and field in which it occurs

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an arrow prompt (=>).  Examples of formats include: TI; TI,AU; BIB,ST;
TI,IND; TI,SO.  You may specify the format fields in any order and the
information will be displayed in the same order as the format
specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR,
FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC
to view a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):ibib

L9   ANSWER 1 OF 32  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2005:634307  CAPLUS
DOCUMENT NUMBER:          143:259192
TITLE:                    STAT1 as a new molecular target of anti-inflammatory
                          treatment
AUTHOR(S):                Carcereri de Prati, Alessandra; Ciampa, Anna Rosa;
                          Cavalieri, Elisabetta; Zaffini, Raffaela; Darra,
                          Elena; Menegazzi, Marta; Suzuki, Hisanori; Mariotto,
                          Sofia
CORPORATE SOURCE:         Section of Biochemistry, Department of Neuroscience
                          and Vision, University of Verona, Verona, 37134, Italy
SOURCE:                   Current Medicinal Chemistry (2005), 12(16), 1819-1828
                          CODEN: CMCHE7; ISSN: 0929-8673
PUBLISHER:                Bentham Science Publishers Ltd.
DOCUMENT TYPE:            Journal; General Review
LANGUAGE:                 English
REFERENCE COUNT:          76    THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> d ibib

L9   ANSWER 1 OF 32  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2005:634307  CAPLUS
DOCUMENT NUMBER:          143:259192
TITLE:                    STAT1 as a new molecular target of anti-inflammatory
                          treatment
```

AUTHOR(S): Carcereri de Prati, Alessandra; Ciampa, Anna Rosa; Cavalieri, Elisabetta; Zaffini, Raffaela; Darra, Elena; Menegazzi, Marta; Suzuki, Hisanori; Mariotto, Sofia
CORPORATE SOURCE: Section of Biochemistry, Department of Neuroscience and Vision, University of Verona, Verona, 37134, Italy
SOURCE: Current Medicinal Chemistry (2005), 12(16), 1819-1828
CODEN: CMCHE7; ISSN: 0929-8673
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d 2-10 ibib

L9 ANSWER 2 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:691126 CAPLUS
DOCUMENT NUMBER: 143:318846
TITLE: Green tea polyphenol extract attenuates **ischemia/reperfusion** injury of the gut
AUTHOR(S): Muia, Carmelo; Mazzon, Emanuela; Paola, Rosanna; Genovese, Tiziana; Menegazzi, Marta; Caputi, Achille P.; Suzuki, Hisanori; Cuzzocrea, Salvatore
CORPORATE SOURCE: Department of Clinical and Experimental Medicine and Pharmacology, Torre Biologica, Policlinico Universitario, Messina, 98123, Italy
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology (2005), 371(5), 364-374
CODEN: NSAPCC; ISSN: 0028-1298
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:779941 CAPLUS
DOCUMENT NUMBER: 143:241673
TITLE: Protective effect of **catechin** on **ischemia-reperfusion**-induced renal injury in rats
AUTHOR(S): Singh, Devinder; Chander, Vikas; Chopra, Kanwaljit
CORPORATE SOURCE: Pharmacology Division, University Institute of Pharmaceutical Sciences, Panjab University, Chandigarh, 160014, India
SOURCE: Pharmacological Reports (2005), 57(1), 70-76
CODEN: PRHEDU; ISSN: 1734-1140
PUBLISHER: Polish Academy of Sciences, Institute of Pharmacology
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1101390 CAPLUS
DOCUMENT NUMBER: 144:535
TITLE: Inhibitory effect of **green tea extract** on β-amyloid-induced PC12 cell death by inhibition of the activation of NF-κB and ERK/p38 MAP kinase pathway through antioxidant mechanisms
AUTHOR(S): Lee, Sun Young; Lee, Jae Woong; Lee, Heesoon; Yoo, Han

Soo; Yun, Yeo Pyo; Oh, Ki Wan; Ha, Tae Youl; Hong, Jin Tae
CORPORATE SOURCE: College of Pharmacy, Chungbuk National University, Chungbuk, Cheongju, Heungduk-gu, 361-763, S. Korea
SOURCE: Molecular Brain Research (2005), 140(1-2), 45-54
CODEN: MBREE4; ISSN: 0169-328X
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:203590 CAPLUS
DOCUMENT NUMBER: 140:210831
TITLE: Composition for protecting organ, tissue or cell and utilization thereof
INVENTOR(S): Komeda, Masashi; Hyon, Suong-Hyu; Miwa, Senri
PATENT ASSIGNEE(S): MG Pharmacy Inc., Japan
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004019680	A1	20040311	WO 2003-JP11127	20030829
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003261860	A1	20040319	AU 2003-261860	20030829
EP 1535514	A1	20050601	EP 2003-791435	20030829
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.: JP 2002-255979 A 20020830
WO 2003-JP11127 W 20030829
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:815203 CAPLUS
DOCUMENT NUMBER: 141:288837
TITLE: Epigallocatechin-3-gallate inhibits STAT1 activation and protects cardiac myocytes from **ischemia/reperfusion**-induced apoptosis
AUTHOR(S): Townsend, Paul A.; Scarabelli, Tiziano M.; Pasini, Evasio; Gitti, Gianluca; Menegazzi, Marta; Suzuki, Hisanori; Knight, Richard A.; Latchman, David S.; Stephanou, Anastasis
CORPORATE SOURCE: Medical Molecular Biology Unit, Institute of Child Health, University College London, London, WC1N 1EH, UK
SOURCE: FASEB Journal (2004), 18(13), 1621-1623, 10.1096/fj.04-1716fje
CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER: Federation of American Societies for Experimental

Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:263484 CAPLUS
DOCUMENT NUMBER: 140:332243
TITLE: (-)-**epicatechin** 3-O-gallate ameliorates the damages related to peroxynitrite production by mechanisms distinct from those of other free radical inhibitors
AUTHOR(S): Yokozawa, Takako; Rhyu, Dong Young; Cho, Eun Ju
CORPORATE SOURCE: Institute of Natural Medicine, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan
SOURCE: Journal of Pharmacy and Pharmacology (2004), 56(2), 231-239
CODEN: JPPMAB; ISSN: 0022-3573
PUBLISHER: Pharmaceutical Press
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:674328 CAPLUS
DOCUMENT NUMBER: 142:148419
TITLE: Protective effects of green tea **catechins** on cerebral ischemic damage
AUTHOR(S): Suzuki, Motohisa; Tabuchi, Masaki; Ikeda, Masahiko; Umegaki, Keizo; Tomita, Takako
CORPORATE SOURCE: Graduate School of Health Sciences, University of Shizuoka, Yada, Shizuoka, Japan
SOURCE: Medical Science Monitor (2004), 10(6), BR166-BR174
CODEN: MSMOFR; ISSN: 1234-1010
PUBLISHER: International Scientific Literature, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 9 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:1039130 CAPLUS
DOCUMENT NUMBER: 142:232867
TITLE: Epigallocatechin, a green tea polyphenol, attenuates myocardial **ischemia reperfusion** injury in rats
AUTHOR(S): Aneja, Rajesh; Hake, Paul W.; Burroughs, Timothy J.; Denenberg, Alvin G.; Wong, Hector R.; Zingarelli, Basilia
CORPORATE SOURCE: Department of Pediatrics, Division of Critical Care Medicine, Cincinnati Children's Hospital Medical Center and College of Medicine, University of Cincinnati, Cincinnati, OH, USA
SOURCE: Molecular Medicine (Manhasset, NY, United States) (2004), 10(1-6), 55-62
CODEN: MOMEF3; ISSN: 1076-1551
PUBLISHER: North Shore-Long Island Jewish Research Institute
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 10 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:742540 CAPLUS
DOCUMENT NUMBER: 140:139058
TITLE: Procyanidins from grape seeds protect endothelial cells from peroxynitrite damage and enhance endothelium-dependent relaxation in human artery: new evidences for cardio-protection
AUTHOR(S): Aldini, Giancarlo; Carini, Marina; Piccoli, Angela; Rossoni, Giuseppe; Facino, Roberto Maffei
CORPORATE SOURCE: Istituto Chimico Farmaceutico Tossicologico, University of Milan, Milan, 20131, Italy
SOURCE: Life Sciences (2003), 73(22), 2883-2898
CODEN: LIFSAK; ISSN: 0024-3205
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L9 ANSWER 11 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:811152 CAPLUS
DOCUMENT NUMBER: 142:233011
TITLE: Studies on effects of tea **catechins** on neuron and Ca2+ concentration in the brain of cerebral ischemia and reperfusion rats
AUTHOR(S): Fang, Fang; Cui, Zhiqing; Han, Yongjing
CORPORATE SOURCE: Institute of Materia Medica, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, 100050, Peop. Rep. China
SOURCE: Zhongguo Yaoxue Zazhi (Beijing, China) (2003), 38(12), 917-919
CODEN: ZYZAEU; ISSN: 1001-2494
PUBLISHER: Zhongguo Yaoxue Zazhishe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

L9 ANSWER 12 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:294597 CAPLUS
DOCUMENT NUMBER: 139:143640
TITLE: Protective activity of (-)-**epicatechin** 3-O-gallate against peroxynitrite-mediated renal damage
AUTHOR(S): Yokozawa, Takako; Rhyu, Dong Young; Cho, Eun Ju; Aoyagi, Kazumasa
CORPORATE SOURCE: Institute of Natural Medicine, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan
SOURCE: Free Radical Research (2003), 37(5), 561-571
CODEN: FRARER; ISSN: 1071-5762
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 80 THERE ARE 80 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 13 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:684340 CAPLUS
DOCUMENT NUMBER: 140:246727
TITLE: Protective effects of the green tea polyphenol, (-)-epigallocatechin gallate against **ischemia reperfusion** injury induced by middle cerebral artery occlusion in rats
AUTHOR(S): Choi, Young-Bin; Park, Jeong-Wook; Han, Si-Ryung; Lee, Kwang-Soo; Kim, Beum-Saeng

CORPORATE SOURCE: Department of Neurology, College of Medicine, The Catholic University of Korea, S. Korea
SOURCE: Taehan Sin'gyong Kwahak Hoechi (2003), 21(4), 387-391
CODEN: TSKHC2; ISSN: 1225-7044
PUBLISHER: Korean Neurological Association
DOCUMENT TYPE: Journal
LANGUAGE: Korean

L9 ANSWER 14 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:126725 CAPLUS
DOCUMENT NUMBER: 139:255093
TITLE: Effect of **green tea extracted** polyphenol on **ischemia/reperfusion** injury after cold preservation of rat lung
AUTHOR(S): Omasa, M.; Fukuse, T.; Matsuoka, K.; Inui, K.; Hyon, S. H.; Wada, H.
CORPORATE SOURCE: Department of Thoracic Surgery, Institute for Frontier Medical Sciences, Kyoto, Japan
SOURCE: Transplantation Proceedings (2003), 35(1), 138-139
CODEN: TRPPA8; ISSN: 0041-1345
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 15 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:484552 CAPLUS
DOCUMENT NUMBER: 139:245148
TITLE: Antioxidant nutrients and hypoxia/ischemia brain injury in rodents
AUTHOR(S): Ikeda, Katsumi; Negishi, Hiroko; Yamori, Yukio
CORPORATE SOURCE: School of Human Environmental Sciences, Mukogawa Women's University, Ikebiraki-cho, Nishinomiya, Japan
SOURCE: Toxicology (2003), 189(1-2), 55-61
CODEN: TXCYAC; ISSN: 0300-483X
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 16 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:809927 CAPLUS
DOCUMENT NUMBER: 138:348524
TITLE: Prevention of hepatic **ischemia-reperfusion** injury by **green tea extract**
AUTHOR(S): Zhong, Zhi; Froh, Matthias; Connor, Henry D.; Li, Xiangli; Conzelmann, Lars O.; Mason, Ronald P.; Lemasters, John J.; Thurman, Ronald G.
CORPORATE SOURCE: Departments of Cell and Developmental Biology and Pharmacology, University of North Carolina at Chapel Hill, Chapel Hill, NC, 27599, USA
SOURCE: American Journal of Physiology (2002), 283(4, Pt. 1), G957-G964
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 17 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:153468 CAPLUS
DOCUMENT NUMBER: 139:224361
TITLE: Inhibitory effects of epigallocatechin gallate on apoptosis in human vascular endothelial cells
AUTHOR(S): Choi, Yean-Jung; Choi, Jung-Suk; Lee, Se-Hee; Lee, Yong-Jin; Kang, Jung-Sook; Kang, Young-Hee
CORPORATE SOURCE: Division of Life Sciences, Hallymr University, Chuncheon, 200-702, S. Korea
SOURCE: Han'guk Sikp'um Yongyang Kwahak Hoechi (2002), 31(4), 672-678
CODEN: HSYHFB; ISSN: 1226-3311
PUBLISHER: Korean Society of Food Science and Nutrition
DOCUMENT TYPE: Journal
LANGUAGE: Korean

L9 ANSWER 18 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:586433 CAPLUS
DOCUMENT NUMBER: 137:231947
TITLE: Cardioprotective abilities of white wine
AUTHOR(S): Cui, Jianhua; Tosaki, Arpad; Cordis, Gerald A.; Bertelli, Alberto A. E.; Bertelli, Aldo; Maulik, Nilanjana; Das, Dipak K.
CORPORATE SOURCE: Cardiovascular Research Center, University of Connecticut School of Medicine, Farmington, CT, 06030-1110, USA
SOURCE: Annals of the New York Academy of Sciences (2002), 957(Alcohol and Wine in Health and Disease), 308-316
CODEN: ANYAA9; ISSN: 0077-8923
PUBLISHER: New York Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 19 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:862167 CAPLUS
DOCUMENT NUMBER: 138:368066
TITLE: Protection of hypoxia/reoxygenation injury by green tea supplementation in cultured cardiac cells
AUTHOR(S): Bordoni, Alessandra; Hrelia, Silvana; Angeloni, Cristina; Leoncini, Emanuela; Giordano, Emanuele; Guarnieri, Carlo; Caldarera, Claudio M.; Biagi, Pier L.
CORPORATE SOURCE: Nutrition Research Center (Department of Biochemistry), Alma Mater Studiorum University of Bologna, Bologna, 40126, Italy
SOURCE: Free Radical Research (2002), 36(Suppl. 1), 75-76
CODEN: FRARER; ISSN: 1071-5762
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 20 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:709688 CAPLUS
DOCUMENT NUMBER: 135:251987
TITLE: Compositions suitable for the treatment of damage caused by **ischemia/reperfusion** or oxidative stress
INVENTOR(S): Van Norren, Klaske; Van Hoorn, Eduard Christiaan; Leuvenink, Hendrik Gerrit Derk; Hofman, Zandrie
PATENT ASSIGNEE(S): N.V. Nutricia, Neth.
SOURCE: Eur. Pat. Appl., 15 pp.
CODEN: EPXXDW

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1136073	A1	20010926	EP 2000-201051	20000322
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
WO 2001070248	A1	20010927	WO 2001-NL233	20010322
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1267901	A1	20030102	EP 2001-915929	20010322
EP 1267901	B1	20060125		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003527434	T2	20030916	JP 2001-568445	20010322
AT 316382	E	20060215	AT 2001-915929	20010322
US 2004076692	A1	20040422	US 2003-239429	20030115

PRIORITY APPLN. INFO.: EP 2000-201051 A 20000322
WO 2001-NL233 W 20010322

OTHER SOURCE(S): MARPAT 135:251987
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 21 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:81613 CAPLUS
DOCUMENT NUMBER: 137:210767
TITLE: Protective effects of tea **catechins** against injury in cerebral ischemia and reperfusion in rats
AUTHOR(S): Fang, Fang; Han, Yongjing; Cui, Zhiqing
CORPORATE SOURCE: School of Chemical Engineering, Tianjing University, Tianjing, 300072, Peop. Rep. China
SOURCE: Zhongguo Zhongyao Zazhi (2001), 26(11), 777-780
CODEN: ZZZAE3; ISSN: 1001-5302
PUBLISHER: Zhongguo Yaoxuehui
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

L9 ANSWER 22 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:113317 CAPLUS
DOCUMENT NUMBER: 135:142037
TITLE: Neuroprotective effect of **green tea extract** in experimental **ischemia-reperfusion** brain injury
AUTHOR(S): Hong, J. T.; Ryu, S. R.; Kim, H. J.; Lee, J. K.; Lee, S. H.; Kim, D. B.; Yun, Y. P.; Ryu, J. H.; Lee, B. M.; Kim, P. Y.
CORPORATE SOURCE: National Institute of Toxicological Research, Korea Food and Drug Administration, Seoul, S. Korea
SOURCE: Brain Research Bulletin (2001), Volume Date 2000, 53(6), 743-749
CODEN: BRBUDU; ISSN: 0361-9230
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 23 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:176372 CAPLUS
DOCUMENT NUMBER: 134:361168
TITLE: Effects of buckwheat in a renal **ischemia-reperfusion** model
AUTHOR(S): Yokozawa, Takako; Fujii, Hajime; Kosuna, Kenichi; Nonaka, Gen-Ichiro
CORPORATE SOURCE: Institute of Natural Medicine, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan
SOURCE: Bioscience, Biotechnology, and Biochemistry (2001), 65(2), 396-400
CODEN: BBBIEJ; ISSN: 0916-8451
PUBLISHER: Japan Society for Bioscience, Biotechnology, and Agrochemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 24 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:7986 CAPLUS
DOCUMENT NUMBER: 134:236821
TITLE: Protective effect of **green tea extract** on **ischemia/reperfusion**-induced brain injury in Mongolian gerbils
AUTHOR(S): Hong, J. T.; Ryu, S. R.; Kim, H. J.; Lee, J. K.; Lee, S. H.; Yun, Y. P.; Lee, B. M.; Kim, P. Y.
CORPORATE SOURCE: National Institute of Toxicological Research, Korea Food and Drug Administration, Seoul, Eunpyung-gu, Nokbun-dong, 122-704, S. Korea
SOURCE: Brain Research (2001), 888(1), 11-18
CODEN: BRREAP; ISSN: 0006-8993
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 25 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:725048 CAPLUS
DOCUMENT NUMBER: 132:44494
TITLE: Inhibition of xanthine oxidase by flavonoids
AUTHOR(S): Nagao, Akihiko; Seki, Michiko; Kobayashi, Hidetaka
CORPORATE SOURCE: National Food Research Institute, Ministry of Agriculture, Forestry and Fisheries, Tsukuba, 305-8642, Japan
SOURCE: Bioscience, Biotechnology, and Biochemistry (1999), 63(10), 1787-1790
CODEN: BBBIEJ; ISSN: 0916-8451
PUBLISHER: Japan Society for Bioscience, Biotechnology, and Agrochemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 26 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:406065 CAPLUS
DOCUMENT NUMBER: 133:163655
TITLE: Protective effect of **green tea extract** against reperfusion injury in rats: antioxidant and anti-inflammatory properties

AUTHOR(S): Yagi, Nobuaki; Yoshikawa, Toshikazu; Naito, Yuji; Matsuyama, Kiichi; Tanaka, Yukiko; Ochiai, Jun; Yoshida, Norimasa; Kondo, Motoharu
CORPORATE SOURCE: First Department of Medicine, Kyoto Prefectural University of Medicine, Kyoto, 602-8566, Japan
SOURCE: Journal of Clinical Biochemistry and Nutrition (1999), 27(2), 89-101
CODEN: JCBNER; ISSN: 0912-0009
PUBLISHER: Institute of Applied Biochemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 27 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:645332 CAPLUS
DOCUMENT NUMBER: 129:339851
TITLE: Catecholic iron complexes as cytoprotective superoxide scavengers against hypoxia/reoxygenation injury in isolated hepatocytes
AUTHOR(S): Zhao, Z. Sylvia; Khan, Sumsullah; O'Brien, Peter J.
CORPORATE SOURCE: Faculty of Pharmacy, University of Toronto, Toronto, ON, M5S 2S2, Can.
SOURCE: Biochemical Pharmacology (1998), 56(7), 825-830
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 28 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:798011 CAPLUS
DOCUMENT NUMBER: 130:138668
TITLE: Oral administration of (-)**catechin** protects against **ischemia-reperfusion** -induced neuronal death in the gerbil
AUTHOR(S): Inanami, O.; Watanabe, Y.; Syuto, B.; Nakano, M.; Tsuji, M.; Kuwabara, M.
CORPORATE SOURCE: Department of Radiation Biology, Faculty of Veterinary Medicine, Hokkaido University, Sapporo, 060-0818, Japan
SOURCE: Free Radical Research (1998), 29(4), 359-365
CODEN: FRARER; ISSN: 1071-5762
PUBLISHER: Harwood Academic Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 29 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:644030 CAPLUS
DOCUMENT NUMBER: 130:57065
TITLE: Biodefense against active oxygens and free radicals induced by oxidative stress
AUTHOR(S): Kuwabara, Mikinori; Inanami, Osamu
CORPORATE SOURCE: Laboratory of Radiation Biology, Graduate School of Veterinary Medicine, Hokkaido University, Sapporo, 060, Japan
SOURCE: Biodefence Mechanisms against Environmental Stress (1998), 23-32. Editor(s): Ozawa, Toshihiko; Tatsumi, Kouichi; Hori, Tada-aki. Kodansha: Tokyo, Japan.
CODEN: 66THAU
DOCUMENT TYPE: Conference
LANGUAGE: English

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 30 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1997:599008 CAPLUS
DOCUMENT NUMBER: 127:272641
TITLE: Effects of tea **catechins** on activities of ATPases and MDA content in cerebral **ischemia -reperfusion** rats
AUTHOR(S): He, Bing; Chen, Xiaoxia
CORPORATE SOURCE: Guangdong College Pharmacy, Canton, 510224, Peop. Rep. China
SOURCE: Guangdong Yaoxueyuan Xuebao (1997), 13(2), 94-96
CODEN: GYXUF8
PUBLISHER: Guangdong Yaoxueyuan
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

L9 ANSWER 31 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:710956 CAPLUS
DOCUMENT NUMBER: 126:1132
TITLE: The in vitro antioxidant activity of trilinolein and other lipid-related natural substances as measured by enhanced chemiluminescence
AUTHOR(S): Chan, Paul; Cheng, Juei-Tang; Tsao, Chiung-Wen; Niu, Chiang-Shan; Hong, Chuan-Ye
CORPORATE SOURCE: Inst. of Clinical Medicine, National Yang-Ming Univ., Taipei, Taiwan
SOURCE: Life Sciences (1996), 59(24), 2067-2073
CODEN: LIFSAK; ISSN: 0024-3205
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

L9 ANSWER 32 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:86000 CAPLUS
DOCUMENT NUMBER: 124:250212
TITLE: Effect of flavonoids on the outcome of myocardial mitochondrial **ischemia/reperfusion** injury
AUTHOR(S): van Jaarsveld, H.; Kuyl, J. M.; Schulenburg, D. H.; Wiid, N. M.
CORPORATE SOURCE: Dept. Chem. Pathology, Univ. Orange Free State, Bloemfontein, 9300, S. Afr.
SOURCE: Research Communications in Molecular Pathology and Pharmacology (1996), 91(1), 65-75
CODEN: RCMPE6; ISSN: 1078-0297
PUBLISHER: PJD Publications
DOCUMENT TYPE: Journal
LANGUAGE: English

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